Public Health Reports

VOLUME 54 MARCH 17, 1939 NUMBER 11

IN THIS ISSUE

UNITED STATES TREASURY DEPARTMENT

PUBLIC HEALTH SERVICE, Thomas Parran, *Surgeon General*

DIVISION OF SANITARY REPORTS AND STATISTICS

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It contains (1) current information regarding the prevalence and geographic distribution of communicable diseases in the United States, insofar as data are obtainable, and of cholera, plague, smallpox, typhus fever, yellow fever, and other important communicable diseases throughout the world; (2) articles relating to the cause, prevention, and control of disease; (3) other pertinent information regarding sanitation and the conservation of the public health.

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UNITED STATES GOVERNMENT PRINTING OFFICE : 1939

For sale by the Superintendent of Documents, Washington, D. C.

Price 5 cents. Subscription price $2 a Year

Public Health Reports

Vol. 54 • MARCH 17, 1939 • *No. 11*

PREVALENCE OF COMMUNICABLE DISEASES IN THE UNITED STATES

January 29–February 25, 1939

The accompanying table summarizes the prevalence of eight important communicable diseases, based on weekly telegraphic reports from State health departments. The reports from each State are published in the Public Health Reports under the section "Prevalence of disease." The table gives the number of cases of these diseases for the 4-week period ending February 25, the number reported for the corresponding period in 1938, and the median number for the years 1934–38.

DISEASES ABOVE MEDIAN PREVALENCE

Smallpox.—Although the number of cases (1,554) of smallpox reported for the 4 weeks ending February 25 was only about 70 percent of the number reported during the corresponding period in 1938, it was still approximately 75 percent greater than the 1934–38 median figure for this period. In the North Central region, Indiana reported 449 cases, Iowa 196 cases, and Ohio 134 cases, while in the West South Central region Texas and Oklahoma reported 148 and 134 cases, respectively—more than two-thirds of the total number of cases occurring in those five States. In the Atlantic Coast regions the incidence remains about normal, no cases being reported from the North Atlantic States and only 7 from the South Atlantic States.

DISEASES BELOW MEDIAN PREVALENCE

Influenza.—There was a sharp increase in influenza cases from approximately 13,000 cases during the 4 weeks ending January 29 to approximately 24,000 cases during the 4 weeks ending February 25.[1] The number of cases was almost twice the number recorded for the corresponding period in 1938, but it was about 10 percent below the 1934–38 median figure for this period. Some increase in the number of influenza cases is to be expected, since the disease is usually quite prevalent at this time of the year, and since the highest incidence of

[1] Later reports appear on pp. 445–446.

the season has normally been reached during the period corresponding to the one under consideration. The disease has apparently been most prevalent in the South Atlantic and East North Central regions, with slight increases over the normal expectancy in the North Atlantic and Mountain regions. The West North Central and Pacific regions reported the lowest incidence in recent years, and in the West South Central region the number of cases was slightly below the average incidence.

Number of reported cases of 8 communicable diseases in the United States during the 4-week period Jan. 29–Feb. 25, 1939, the number for the corresponding period in 1938, and the median number of cases reported for the corresponding period 1934–38 [1]

Division	Current period	1938	5-year median	Current period	1938	5-year median	Current period	1938	5-year median	Current period	1938	5-year median
	Diphtheria			Influenza [2]			Measles [3]			Meningococcus meningitis		
United States [1]	1,994	2,436	2,436	23,994	12,990	28,549	53,546	131,829	91,667	227	378	525
New England	39	36	53	122	76	76	6,201	2,410	5,686	12	12	12
Middle Atlantic	331	414	394	840	131	220	5,590	42,069	11,383	50	60	60
East North Central	411	553	531	5,016	397	1,212	5,799	51,204	10,255	19	38	98
West North Central	168	196	222	793	836	1,285	8,274	6,372	6,372	13	28	54
South Atlantic	383	397	422	9,184	2,995	8,337	9,968	14,954	9,232	41	79	93
East South Central	139	178	192	2,196	1,996	3,630	1,494	8,767	4,589	51	98	92
West South Central	299	400	400	4,322	5,299	5,058	2,117	2,338	1,667	20	38	43
Mountain	86	116	71	1,170	585	812	3,251	2,628	2,628	11	9	23
Pacific	138	146	176	351	675	2,390	10,852	1,087	2,764	10	16	18
	Poliomyelitis			Scarlet fever			Smallpox			Typhoid and paratyphoid fever		
United States [1]	66	89	80	22,169	24,290	26,877	1,554	2,241	883	433	523	521
New England	0	2	2	1,539	1,893	1,585	0	0	0	12	16	14
Middle Atlantic	7	5	6	5,100	5,682	6,539	0	0	0	50	54	55
East North Central	9	9	11	8,659	8,245	9,057	749	503	179	52	86	73
West North Central	4	4	6	2,507	3,765	3,765	254	661	413	18	36	36
South Atlantic	17	17	12	1,087	1,034	1,034	7	11	5	74	85	85
East South Central	14	19	9	623	615	614	42	193	9	47	37	58
West South Central	6	9	6	764	844	654	273	268	185	144	153	141
Mountain	4	8	3	586	897	897	134	193	125	13	27	19
Pacific	5	16	16	1,804	1,315	1,406	95	412	148	23	29	29

[1] 48 States. Nevada is excluded and the District of Columbia is counted as a State in these reports.
[2] 44 States and New York City.
[3] 46 States. Georgia and Mississippi are excluded.

Measles.—While the number of cases of measles rose from approximately 37,000 for the preceding 4-week period to more than 53,000 for the 4 weeks ending February 25, the incidence was only about 45 percent of that for the corresponding period in 1938 and was approximately 38,000 cases less than the 1934–38 average number for this period. The incidence in relation to recent years was especially high in the Pacific area, considerably above normal in the West North Central, West South Central, and Mountain States, and about 10 percent above the seasonal average in the New England and South Atlantic regions. On the other hand, the Middle Atlantic and East North Central areas reported only about one-half of the 5-year average

number of cases for this period, and in the East South Central area the number of cases was only about one-third of the 1934–38 median figure.

Poliomyelitis.—The incidence of poliomyelitis was about 20 percent below the average incidence for this season of the year. For the 4 weeks ending February 25 the number of reported cases totaled 66, as compared with 89, 80, and 66 for the corresponding period in 1938, 1937, and 1936, respectively. In the South Atlantic and East South Central regions the incidence was slightly above the seasonal average, while in the Pacific area the number of cases was relatively low; other regions reported about the normal seasonal incidence.

Diphtheria.—For the 4 weeks ending February 25 there were 1,994 cases of diphtheria reported, as compared with 2,436, 2,069, and 2,369 for the corresponding period in 1938, 1937, and 1936, respectively. With the exception of a slight interruption in 1938, there has been a steady decline in the incidence of diphtheria. The current incidence is the lowest for this period in the 11 years for which these data are available; in 1929 the cases for this period totaled approximately 6,100 cases.

Typhoid fever.—The typhoid fever incidence was relatively low. The number of cases (433) reported for the current period was only about 80 percent of the average incidence for recent years. In the West South Central region the number of cases was approximately the same as the 1934–38 average figure for this period, but in all other regions the incidence was below the seasonal expectancy.

Scarlet fever.—The scarlet fever incidence was comparatively low—22,169 cases as compared with approximately 24,000 cases in 1938, and an average of approximately 27,000 cases for the years 1934–38. Very significant decreases in the numbers of cases as compared with the experience of recent years were reported from the Middle Atlantic, North Central, and Mountain regions. For the country as a whole the number of cases reported during the current period was the lowest reported for the corresponding period in 10 years.

Meningococcus meningitis.—For the 4 weeks ending February 25 there were 227 cases of meningococcus meningitis reported, as compared with 378, 678, and 800 for the corresponding period in 1938, 1937, and 1936, respectively. Each geographical division of the country shared in the favorable situation, the incidence in each region, except the New England, being considerably below the normal seasonal occurrence of this disease. For the country as a whole the current incidence is the lowest since 1934, when a total of 227 cases was then reported for the corresponding period.

MORTALITY, ALL CAUSES

The average mortality rate from all causes in large cities for the 4 weeks ending February 25, based on data received from the Bureau

of the Census, was 13.2 per 1,000 inhabitants (annual basis). The rates for the corresponding periods in the years 1938, 1937, and 1936 were 12.0, 14.3, and 13.8, respectively. While the current rate is considerably above that of last year, it is lower than the rates for the corresponding period of the two preceding years. The low rate of 1938 was no doubt largely due to the low incidence of influenza during the winter months. While the reported influenza incidence is not relatively high this year for the country as a whole, it might be a contributing factor to the increased death rates in some large cities located in regions from which large numbers of influenza cases have been reported.

MOUTH LESIONS ASSOCIATED WITH DIETARY DEFICIENCIES IN MONKEYS

By N. H. Topping, *Assistant Surgeon*, and H. F. Fraser, *Passed Assistant Surgeon, United States Public Health Service, National Institute of Health*

INTRODUCTION

The etiological factors responsible for some of the various types of soft tissue lesions occurring in the oral cavity are not clearly understood. The infectious theory, with the spirals and fusiforms implicated as the causative agents, has been investigated by numerous workers (*1*) since the original papers of Plaut (*2*) and Vincent (*3*). Recently, in aphthous stomatitis, still another agent has been suggested—the virus of herpes simplex (*4*). This type of stomatitis has yielded the virus by inoculations into rabbits' corneas, and the recovered animals were subsequently immune to inoculations with a known strain of herpes simplex virus (*4*). A vesicular stomatitis has been described in animals and the virus has been recovered (*5*). The recovery of these viruses from vesicular lesions has placed this type of infectious agent on a sound basis as the etiological factor in these lesions.

The etiology of the periodontal diseases, including gingivitis, chronic, subacute and acute Vincent's infections, and pyorrhea alveolaris, remains for the most part unsettled. For some time various dietary deficiency diseases in their milder forms have been suggested as possible factors in the production of these conditions. The effect on the oral tissues of dogs depleted of vitamin A was studied by Mellanby (*6*); the effects of vitamin C deficiency have been studied both clinically (*7*) and experimentally (*8*); and recently certain components of the B_2 complex have been implicated, both from experimental work (*9*) and from clinical observations (*10*). However, many of these were supplementary observations made during the course of experi-

ments designed for other purposes, and therefore were not adequately controlled from the viewpoint of a study planned primarily for an investigation of stomatitis. With this in mind, an experiment was designed to determine the occurrence of oral pathology in monkeys on selected dietary deficiencies.

EXPERIMENTAL

Selection of animals and preliminary observations.—Macacus rhesus monkeys were selected for this work because their dental formula is similar to that of man, and because this primate is the only species except man known to require both vitamin C and nicotinic acid, two of the nutritional factors which it was desired to test.

Fifty monkeys were examined clinically, with special attention to the condition of the oral cavity. In addition, a series of stool cultures, one complete blood count, and one blood serum cevitamic acid determination [1] were made on each prospective experimental animal.

Following such initial observations, 14 used monkeys which had been maintained on a stock diet and 26 fresh stock monkeys were selected and placed in individual screen-bottom cages in 2 rooms connected by an open doorway and 1 large open window.

During a subsequent 3-week standardization period on stock diet 512, 2 of the original 40 animals were discarded because of anemia and replaced with animals observed in a preliminary manner for only 2 days. The monkeys were then divided into groups, being equalized as evenly as possible, taking into account age as estimated from the teeth,[2] sex, the results of stool cultures, and whether or not they were used or fresh stock animals.

Diets served monkeys.—The various groups were then placed on control and selected deficiency diets. The details of these diets, their preparation, and concurrent check assays of the monkey diets in rats are presented below.

Composition of monkey diets 482 and 483 with supplements

Nutrients	Diet number	
	482	483
	Grams	*Grams*
Corn meal [1]	320	350
Cowpeas [1]	50	50
Cottonseed oil [1]	50	50
Sucrose [1]	45	45
Salt mixture (Osborne & Mendel) [1]	24	24
Casein, leached	60	86
Yeast, dried brewers'	60	----------

[1] These items are stirred into water in a double boiler of enamelware and cooked for about 1½ hours. Then the other ingredients are well stirred in, the completed diet is dried in cakes for 24 hours and fed so that each monkey receives approximately 800 calories per day.

[1] We are indebted to Dr. Mary E. Reid for the blood serum cevitamic acid determinations throughout the course of this study.

[2] Age estimated from "The Anatomy of the Monkey," by Carl Hartmann.

Supplements

Diet 482

A. Control animals:
1. Vitamin A (carotene in cottonseed oil (S. M. A. Corp.)). 1 cc. (7,500 units) given by mouth once a week to each monkey.
2. Vitamin D (crystalline in propylene glycol (Winthrop Chemical Co.)). 0.33 cc. (13,300 units) given by mouth once a week to each monkey.
3. Vitamin C (crystalline ascorbic acid (Merck) neutralized with sodium bicarbonate). 30 mg. 5 days a week and 60 mg. 1 day a week given by intramuscular injection.

B. Deficient animals:
Vitamin A, D, or C deficient animals received the vitamins described for the controls with one vitamin deleted according to the deficiency desired.

Diet 483

A. Control animals:
1. Riboflavin, synthetic (Winthrop Chemical Co.). 0.5 mg. given intramuscularly once a week in distilled water.
2. Nicotinic acid, 20 mg. given once a week, dissolved in distilled water.
3. Vitamins A, D, and C were given as outlined for diet 482.

B. Deficient animals:
1. B_1 complex deficient animals did not receive riboflavin or nicotinic acid. (When ingredients comparable to those of basal diet 483 are assayed on rats for B_4 (*11*), adequate amounts are present. According to work on chicks (*12, 13*) and rats (*14*), it probably contains an insufficient amount of the filtrate factor. No filtrate factor concentrate was supplied in these experiments).
2. Nicotinic acid deficient animals received a supplement of flavin as described for controls.
3. Flavin deficient animals received a supplement of nicotinic acid as described for controls.
4. Vitamins A, D, and C were given as outlined for diet 482.

Composition and schedule of monkey diet 495a

Monday	Tuesday	Wednesday	Thursday	Friday	Saturday	Sunday
Irish potatoes (cooked)	Bread (whole wheat)	Carrots (raw)	Carrots (cooked)	Irish potatoes (cooked)	Bread (whole wheat)	Bananas
Milk	Milk	Milk	Milk	Milk	Milk	Two eggs (cooked)
Lettuce	Bananas	Bananas	Lettuce	Cabbage (cooked)	Cabbage (raw)	
	One egg (cooked)	Lettuce				

Approximately 1,000 calories were provided for each monkey daily. Since they ate about 800 calories daily, this permitted some freedom of choice in quantity of individual foods consumed. The monkeys ate all of the above diet very well except the cooked carrots.
The food was prepared as follows:

Milk was made from whole milk powder daily and the equivalent of about 250 cc. of whole milk was given daily except Sunday.
Eggs were boiled approximately 5 minutes.
Excessive cooking of vegetables was carefully avoided.

Composition of monkey stock diet 512

Each monkey received 6 days a week approximately 125 gm. of whole wheat bread, 250 cc. of whole milk, and three bananas. One day a week each monkey received two hard-boiled eggs.

Concurrent check assays of monkey diets in rats.—A group of 75 rats, including males and females, ranging in age from 21 to 25 days, were used to test each diet as prepared for monkeys, except the diet deficient in vitamin C and diet 495a composed of natural foods. They were observed for rate of growth, symptoms, ability to reproduce, and success in lactation. Rats on control artificial diet 482 with supplements of vitamins A and D grew well and were successful in reproduction and lactation. Rats on basal diet 482 plus vitamin D but minus vitamin A grew rather poorly and did not reproduce; but since they maintained their weight fairly well and did not develop xerophthalmia, the presence of some vitamin A in the basal diet was suggested. Rats on B_1 complex control diet 483 with supplements of vitamins A, D, nicotinic acid, and flavin grew quite well, reproduced, and some were partially successful in lactation. Two red blood cell and two white blood cell counts were made at varying intervals from five rats in each of the four groups of the B_1 complex and showed no definite evidence of anemia or leucopenia. (The satisfactory response of control B_1 complex rats on basal diet 483, plus supplements of vitamins A, D, nicotinic acid and flavin, is noteworthy in view of the poor response of monkeys on a similar diet.)

Routine observations on monkeys.—Once weekly, or more frequently when necessary, routine observations were made on each animal's weight, general condition, and particularly the condition of the oral cavity. Determinations of hemoglobin content and total white and red cell counts were made less frequently. In addition, blood serum cevitamic acid [3] determinations were made to ascertain the extent of depletion in deficient animals and the adequacy of the vitamin C supplement.[4]

Smears were made from the gums of all the animals every other week. The same method for making these smears was followed throughout, and consisted of passing a small cotton swab over the gums, several millimeters away from the cervical margin. These swabs were streaked on slides, stained with gentian violet, studied under oil immersion at once, and then saved for final study when the entire set was completed upon the death of the monkey.

Stool cultures were made every 2 weeks on all the monkeys and more frequently on those developing diarrhea. Both eosin methylene blue and desoxycholate citrate agar were used as primary isolation media and *Shigella paradysenteriae* (Flexner) was recovered many times. Blood cultures were taken on two of the monkeys developing noma, and both were negative.

In the planning of the experiment it was considered desirable that all animals proceed to natural death without treatment,[5] and this plan was rigidly adhered to, with the following exceptions: (1) Animals obviously *in extremis* were killed with chloroform in order that postmortem autolysis would not interfere with the pathological studies. (2) Seven control animals and one A-deficient animal were killed with chloroform after about 210 days in order to terminate the experiment.

Photographs were taken of the mouths of all the monkeys before the experimental diets were begun, of those in which pathology was observed, and, finally, of most of the animals at death. These photographs were carefully reviewed in evaluating the clinical progression of the lesions, and particularly in weighing the severity of lesions at death.

Since it is impracticable to publish the series of photographs for each animal, typical examples have been selected of the mouths of two normal animals, following 209 and 210 days on the control diets, and other pictures demonstrating each type of lesion (as listed in table 1) observed at death. These photographs are presented in figures 1 to 8, inclusive, and illustrate the descriptive terms employed.

A routine autopsy was performed and the various tissues were preserved for histopathological studies by Passed Assistant Surgeon T. H. Tomlinson, of the Division of Pathology, National Institute of

[3] Pure synthetic vitamin C.

[4] A dose of 30 mg. of vitamin C given 5 days a week and 60 mg. 1 day a week was considered sufficient, since the average blood serum cevitamic acid of monkeys on the artificial diet plus vitamin C was higher than that of monkeys on diet 495a composed of foods with a high vitamin C content.

[5] A preliminary experiment was conducted on 20 monkeys using the same basal diets reported in this experiment, but with the onset of deficiency symptoms, specific therapy was instituted.

TABLE 1.—*Summarization of pertinent data*

Diet No.	Vitamin deficiency	Monkey number	Sex	Estimated age in months at beginning of experiment	Mouth lesions developed after—days	Description of mouth lesions when first noted	Progress of lesions	Smears from gums increase in—		Description of mouth lesions at death								Survival time in days	Cause of death and autopsy findings
								Fusiform	Spirals	Soft tartar	Inflammation	Localized recession	Gingivitis	Necrosis of interdental papillae	Necrotic gingivitis	Ulcerations in buccal mucosa	Noma		
495a[1]	None (controls)	34	♀	40	0	None	None			0	0	0	0	0	0	0	0	210	Chloroform.
		60	♀	31	0	do	do			0	0	0	0	0	0	0	0	211	Do.
		61	♀	33	141	Some localized gingivitis	Chronic gingivitis→subsided		±	2+	0	1+	0	0	0	0	0	209	Chloroform; tuberculosis.
		65	♂	27	0	None	None			0	0	0	0	0	0	0	0	210	Chloroform.
482	None (controls receiving vitamins A, C, and D)	39	♂	38	84	Slight localized gingivitis	Cleared	2+ Temporary	3+ Temporary	0	0	0	0	0	0	0	0	210	Chloroform.
		41	♀	33	0	None	None			0	0	0	0	0	0	0	0	211	Do.
		50	♀	33	63	Slight gingivitis in upper left premolar and molar area.	Cleared	1+	1+	1+	0	0	0	0	0	0	0	124	Not definitely established.
		55	♀	49	0	None	None	2+	3+	0	0	0	0	0	0	0	0	209	Chloroform.
		58	♀	33	84	Very slight gingivitis in upper right and left premolar areas.	Cleared			1+	0	0	0	0	0	0	0	141	Tuberculosis.
		69	♂	40	13	Slight gingivitis with some localized recession.	Doubtful progression			2+	2+	1+	1+	1+	0	0	0	120	Do.
		73	♀	38	0	None	None			0	0	0	0	0	0	0	0	7	Acute gastro-enteritis of unexplained origin
		79	♂	26	84	Very slight gingivitis in upper premolar region.	Cleared			1+	0	0	0	0	0	0	0	145	Tuberculosis.
	A deficient	36	♀	26	42	Slight localized gingivitis	Cleared			0	0	0	0	0	0	0	0	211	Chloroform.
		46	♂	38	138	Slight gingivitis with hard tartar.	No progression			3‡	0	0	0	0	0	0	0	194	Tuberculosis.
		51	♂	42	168	Very slight gingivitis	Very little progression			2+	2+	2+	1+	1+	0	0	0	164	Do.
		68	♂	26	0	None	None			0	0	0	0	0	0	0	0	82	Not definitely established.

	D deficient	43	♂	47	50	Very slight hypertrophic gingivitis.	Gingivitis → recession → localized necrosis.	2+	2+	3+	2+	3+	2+	2+	1+	0	0	63	Perforation of gut.
		44	♀	26	49	Early necrosis of interdental papillae.	Died before progress could be noted.	----------	2	2+	2+	2+	2+	2+	1+	0	0	50	Tuberculosis.
		53	♂	----	49	Very slight gingivitis	Some progression	(±)	(±)	2+	1+	0	1+	0	0	0	0	101	Do.
		75	♂	35	63	Soft tartar and some gingivitis at cervical margin.	Further accumulation of tartar and localized separation of gum at cervical border.	----------	----------	2+	2+	1+	1+	0	0	0	0	127	Do.
482	C deficient	40	♂	26	15	Moderate redness in upper right and left premolar region.	Generalized redness→hemorrhage→ulcerations→necrosis.	3+	3+	3+	4+	4+	4+	4+	4+	0	0	111	Scurvy; tuberculosis.
		47	♂	33	15	Very slight redness in upper right and left molar areas.	do	2+	3+	3+	4+	4+	4+	4+	4+	0	0	75	Scurvy.
		57	♀	38	43	Some gingivitis and slight hemorrhage at cervical margin.	Some progression	----------	----------	2+	3+	3+	2+	2+	0	0	0	120	Scurvy; tuberculosis.
		71	♀	49	14	Some gingivitis with diffuse reddening of gums at cervical margin.	Generalized redness→hemorrhage→detachment of gums →necrosis.	3+	2+	1+	3+	4+	4+	3+	2+	0	0	75	Scurvy.
483	None [2] (controls receiving flavin and nicotinic acid	30	♀	26	63	Areas of hemorrhagic gingivitis.	Gingivitis → generalized redness at cervical margin→ necrosis.	----------	----------	0	4+	3+	4+	4+	4+	0	0	80	Not definitely established.
		48	♂	26	72	Slight localized gingivitis.	Very little progression	----------	----------	1+	2+	0	2+	0	0	0	0	122	Tuberculosis.
		56	♀	49	42	Generalized redness of gums with some hypertrophy.	Redness→hypertrophy→localized necrosis.	2+	2+	2+	3+	2+	3+	1+	1+	0	0	146	Do.
		63	♂	33	22	Areas of hemorrhagic gingivitis.	Hemorrhagic gingivitis → necrosis of gums→extension to mouth mucosa→noma.	3+	3+	1+	4+	2+	4+	2+	4+	4+	4+	36	Noma.
	Flavin [3] deficient	45	♀	49	0	None	None	----------	----------	0	0	0	0	0	0	0	0	27	Tuberculosis.
		49	♂	33	36	Moderate redness at cervical margin.	Very little progression	2+	2+	0	3+	0	3+	2+	1+	0	0	49	Bacillary dysentery.
		52	♂	31	63	Generalized diffuse redness of gums.	Redness→hemorrhagic gingivitis→necrotic gingivitis→ extension to cheeks→bilateral noma.	1+	2+	2+	4+	3+	4+	4+	4+	4+	4+	84	Noma; tuberculosis.
		78	♂	26	0	None	None	----------	----------	2+	0	0	0	0	0	0	0	59	Tuberculosis.

See footnotes at end of table.

TABLE 1.—*Summarization of pertinent data*—Continued

Diet No.	Vitamin deficiency	Monkey number	Sex	Estimated age in months at beginning of experiment	Mouth lesions developed after — days	Description of mouth lesions when first noted	Progress of lesions	Smears from gums increase in—		Description of mouth lesions at death								Survival time in days	Cause of death and autopsy findings
								Fusiform	Spirals	Soft tartar	Inflammation	Localized recession	Gingivitis	Necrosis of interdental papillae	Necrotic gingivitis	Ulcerations in buccal mucosa	Noma		
483	Nicotinic acid[2] deficient	38	♀	26	35	Generalized redness of gums.	Redness→necrosis of interdental papillae→necrotic gingivitis.	----------	----------	2+	3+	3+	3+	3+	2+	0	0	77	Not definitely established.
		59	♂	26	72	Slight localized gingivitis.	Very little progression	----------	----------	0	2+	0	0	0	0	1+	0	85	Tuberculosis.
		67	♂	33	0	None	None	----------	----------	0	0	0	0	0	0	0	0	38	Possibly bacillary dysentery.
		72	♂	39	13	Hemorrhage from gums	Hemorrhage → hemorrhagic gingivitis→necrosis of gums →extension to cheeks.	2+	2+	3+	4+	3+	4+	4+	4+	4+	0	69	Bacillary dysentery.
	Flavin[2] and nicotinic acid deficient	35	♂	48	26	Faint red line at cervical margin.	Severe gingivitis→necrosis of gums→extension to buccal mucosa→noma.	2+	4+	2+	4+	4+	4+	4+	4+	4+	4+	134	Noma; tuberculosis.
		62	♀	36	77	Gingivitis at cervical margin.	Gingivitis→necrosis of interdental papillae → necrotic gingivitis→extension of buccal mucosa.	----------	----------	3+	4+	2+	4+	4+	4+	3+	0	103	Not definitely established.
		64	♂	38	28	Generalized redness hypertrophy of upper interdental papillae.	Redness→superficial ulcerations in sulci→hemorrhagic gingivitis→necrosis.	2+	2+	2+	4+	3+	4+	3+	3+	0	0	80	Tuberculosis.
		77	♂	26	35	Redness at cervical margin.	Redness→necrosis of some interdental papillae→hemorrhagic gingivitis.	2+	2+	2+	2+	2+	2+	2+	2+	0	0	90	Do.

[1] Diet 495a is composed of natural foods.

[2] The control monkeys in the vitamin B_2 complex group with a basal diet containing vitamin B_1 and receiving supplements of nicotinic acid flavin vitamins A, D, and C responded in such a manner that they may have required one or more additional factors (contained in the yeast of diet 482) for normal nutrition. (See supplements diet 483.)

0=None.
1+=Slight.
2+=Mild.
3+=Moderate.
4+=Severe.
±=Hard tartar.

Health. The pathological findings are made the basis of a concurrent report in this issue of the Public Health Reports.[6] In the great majority of animals our clinical impression of the oral lesions was confirmed by these pathological studies.

The records of all the experimental animals and a summary of pertinent data is presented in table 1.

DISCUSSION OF TABLE 1

General.—Observing the table as a whole, it is at once evident that the control animals on both stock diet 495a and control diet 482 failed to develop as many or as severe mouth lesions as those animals on deficient diets. Individual monkeys, however, in all deficient groups show considerable variation regarding the extent of oral pathology.

Although table 1 shows tuberculosis as a significant cause of death in only 20 monkeys, gross or microscopic lesions typical of tuberculosis were found at autopsy in 26 out of 40 animals. However, this does not appear to invalidate observations on the development of mouth lesions, since table 1 shows that there is apparently no correlation between the incidence of clinical tuberculosis and mouth pathology.

Control monkeys on stock diet 495a.—All of these four animals lived to the termination of the experiment on the two hundred and tenth day. Three of the four animals, 34, 60, and 65, had no lesions of the oral cavity at any time, and at death their gums were in as good condition as, or even better than, at the beginning of the experimental period. One monkey, 61, developed a mild, localized gingivitis, which subsided to the mild chronic form. There was no progression of the lesions. He was diagnosed clinically as having tuberculosis on August 9, 1938, 2 months before the experiment was terminated.

Control monkeys on diet 482 with appropriate supplements.—There were eight animals in this group. One of these, monkey 73, will not be considered, as he died on the seventh day of the experiment. Three of the remaining seven animals were killed to terminate the experiment on the two hundred and tenth day. The average survival time for the group was 165+[7] days. Two monkeys, 41 and 55, at no time had any lesions in the oral cavity. Four, 39, 50, 58, 79, had at some time very slight, transient gingivitis, which cleared before death. Three of these, 50, 58, and 79, had a slight amount of soft tartar along the cervical gum margin at death. Only one monkey out of the seven, 69, developed a moderate degree of inflammation about the gingival margin, which became chronic before death. This

[6] The next article in this issue.

[7] The plus mark following days of survival means that one or more monkeys in the group were chloroformed in order to terminate the experiment, and so it is impossible to calculate the complete survival time of the group.

animal had positive stool cultures for *S. paradysenteriae* (Flexner) before the experimental diet started. On several occasions there was blood and mucus in his stools, and during the experimental period practically all of his stools were positive for Flexner organisms. Paralysis of an undetermined cause developed in the left leg of the animal about 1 month after the experiment was started and continued until death. Gross lesions of a widely disseminated tuberculosis were found at autopsy. All of these facts make it extremely difficult to evaluate this monkey's condition.

"A" deficient monkeys on diet 482.—Of the four monkeys on diet 482 deficient in vitamin A, one was alive at the termination period of 210 days. The average survival period for the group was 162+ days, which compares favorably with the control group on the artificial diet. A simultaneously conducted rat experiment and observations of other workers on cowpeas (*15*) suggest that although this diet contains some vitamin A, it is nevertheless deficient in this respect. One animal in this group, 68, at no time developed lesions of the oral cavity. Two monkeys, 36 and 46, manifested some transient gingivitis which cleared up by the time of death. One animal, 46, developed hard tartar on his incisor teeth, the only 1 of all 40 animals upon which this was observed. The remaining monkey in the group, 51, showed a chronic gingivitis which was present at death.

"D" deficient monkeys on diet 482.—Of the four monkeys on diet 482 deficient in vitamin D, none was alive on the two hundred and tenth day. The average survival time for the group was 85 days. All four animals in this group developed some pathology of the gums. Monkeys 53 and 75 developed only a slight gingivitis. Monkeys 43 and 44 developed a gingivitis which proceeded to necrosis of some of the interdental papillae and finally to a slight generalized necrotic gingivitis.

"C" deficient monkeys on diet 482.—Of the four monkeys on diet 482 deficient in vitamin C, none was alive on the two hundred and tenth day. The average survival time for the group was 95 days. Three of the four monkeys developed a severe gingivitis, while the other, 57, had only a mild gingivitis. The latter monkey received 60 mg. of cevitamic acid by error shortly after manifesting gum lesions, which then cleared and did not recur with the same severity as those of the other three members of the group. In this monkey the lesions progressed only to a necrosis of the interdental papillae, while in the other three, 40, 47, and 71, all developed a generalized necrotic gingivitis, which in two, 40 and 47, was extremely severe. The diagnosis of scurvy was confirmed at autopsy by the findings of hemorrhage into the joints, subperiosteum, and in one instance, into the gastrointestinal tract.

FIGURE 1.—Postmortem photograph Monkey 55, illustrating normal gingiva and mucosa. (Artificial control diet.)

FIGURE 2.—Postmortem photograph Monkey 65, illustrating normal gingiva and mucosa. (Stock diet.)

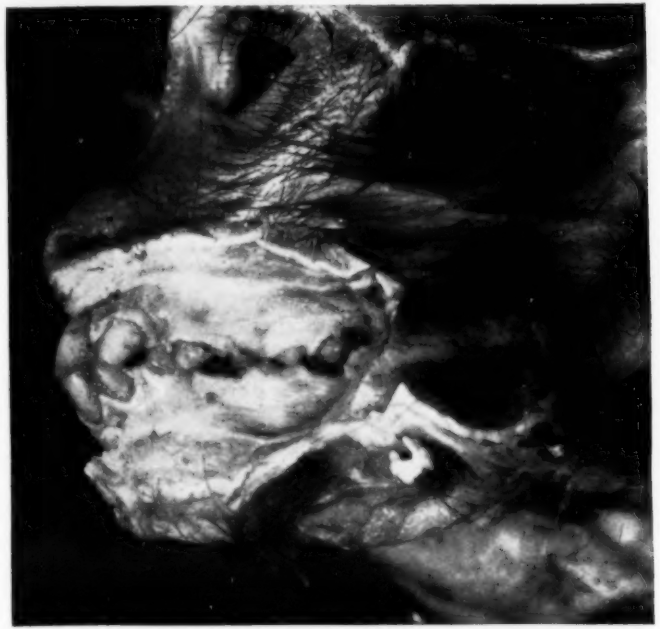

FIGURE 3.—Postmortem photograph Monkey 51, illustrating 2+ soft tartar, 2+ inflammation, 2+ localized recession, 1+ necrosis of interdental papillae, and 1+ necrotic gingivitis. (A deficient diet.)

FIGURE 4.—Postmortem photograph Monkey 47, illustrating 3+ soft tartar, 4+ inflammation, 4+ localized recession, 4+ gingivitis, 4+ necrosis of interdental papillae, and 4+ necrotic gingivitis. (C deficient diet.)

FIGURE 5.—Postmortem photograph Monkey 40, illustrating 3+ soft tartar, 4+ inflammation, 4+ localized recession, 4+ gingivitis, 4+ necrosis of interdental papillae, and 4+ necrotic gingivitis. (C deficient diet.)

FIGURE 6.—Postmortem photograph Monkey 72, illustrating 3+ soft tartar, 4+ inflammation, 3+ localized recession, 4+ gingivitis, 4+ necrosis of interdental papillae, 4+ necrotic gingivitis, and 4+ ulcerations of buccal mucosa. (Note arrows.) (Nicotinic acid deficient diet.)

FIGURE 7.—Postmortem photograph Monkey 35, illustrating 2+ soft tartar, 4+ inflammation, 4+ localized recession, 4+ gingivitis, 4+ necrosis of interdental papillae, 4+ necrotic gingivitis, 4+ ulcerations in buccal mucosa, and noma. (Flavin and nicotinic acid deficient diet.)

FIGURE 8.—Photograph taken shortly before death of Monkey 63, illustrating a case of noma. (B_2 complex control diet, containing flavin and nicotinic acid.)

B_2 complex deficient monkeys.—It was hoped at the beginning of the experimental period that some light would be thrown on the B_2 complex requirements of the monkey. However, the development of intercurrent infections so complicated the results that little knowledge could be gleaned of the requirements of this species. Nevertheless, certain characteristic mouth lesions developed in this group with sufficient regularity to be significant, more especially since this type of lesion was not observed in any of the other groups.

There were 4 groups of 4 monkeys each, making a total of 16, on some phase of the B_2 complex deficiency. None of these monkeys was alive at the end of the experimental period of 210 days. The average survival time for the 4 groups was 73 days. All of the 16 developed a severe watery diarrhea, yet in only 2 of these was a possible etiological cause demonstrated. In many of these monkeys some anemia associated with a moderately severe leucopenia developed which was not observed in other groups of monkeys. On B_2 complex deficient diets, anemia (*16*) (*20*), leucopenia (*9c*) (*20*), and diarrhea (*9a*) (*20*) have been observed in monkeys; and diarrhea has been described in other species (*17*).

Twelve of the 16 animals developed gingivitis; 11 had necrosis of the interdental papillae; 10 had generalized necrotic gingivitis, 6 developed ulcerative lesions in the buccal or labial mucosa where it came in contact with the gingival margins; and, finally, 3 in the group progressed to complete gangrenous necrosis of the cheek which was similar in appearance to the lesions described in human beings, as "noma." Only 2 monkeys, 45 and 67, had no lesions of the oral cavity at any time. Both of these died very early of an intercurrent infection in 27 and 38 days, respectively.

Ulcerative lesions of the gums have been described recently in monkeys on B_2 complex deficiencies (*9a*) (*9c*) (*16*) (*20*). However, to our knowledge, the extension of these lesions to the buccal or labial mucosa has not been recorded. These mucosal lesions observed in monkeys vary considerably from those described in canine black-tongue (*18*).

From our data it would appear that neither nicotinic acid nor flavin, either individually or together, would prevent these lesions from developing under the conditions of this experiment; however, it must be pointed out that the groups of monkeys receiving either or both of these substances survived longer than the group of monkeys receiving neither.

Sebrell and coworkers (*19*) have maintained dogs in an excellent condition for a period of 6 months on a basic diet comparable to diet 483 of this experiment when the dogs' diet was supplemented with cod liver oil, nicotinic acid, and flavin. In a concurrent rat experiment, here reported, using diet 483 and supplements of A, D, nicotinic

acid, and flavin, a fair rate of growth and successful reproduction have been obtained. The poor response, therefore, of the control monkeys in the B_2 complex group receiving diet 483, plus supplements of A, D, nicotinic acid, and flavin, is noteworthy. These observations and those of Day, Langston, and Darby (*9c*) in monkeys suggest that our control B_2 complex diet, as compared with the same diet with yeast added, was still deficient for normal nutrition in the monkey.

A significant observation is that only the monkeys on the B_2 complex deficiencies developed primary ulcers at sites where the teeth and buccal mucosa approximate, yet the C-deficient group had just as severe or even more extensive lesions in the gums proper.

Results of smears from the gums.—Spirals and fusiforms were present in varying numbers in the smears from all the monkeys during the preliminary observation period. Some of the monkeys (41, 44, 45, 47, 51, 55, 59, 61, 67, 69, 71, 72, 73, 77, 78, 79) had rather large numbers of these organisms at this time, while the others had but few. Table 1 shows that there is apparently no correlation between the original number of organisms and subsequent development of mouth lesions. The data regarding the increase of spirals and fusiforms refers to gum smears made from the entire mouth. It shows that as ulcerative lesions develop in the gums the number of spirals and fusiforms increases to some degree; but this was not consistently observed. However, when smears were made directly from ulcerative lesions instead of the entire mouth large numbers of spirals and fusiforms could be demonstrated in almost every instance.

Condensations of table 1.—For purposes of clearness and emphasis, the data of table 1 have been condensed somewhat in tables 2 and 3.

TABLE 2.—*Survival time in days of monkeys on various diets tested*
(Condensation of data presented in table 1)

Group	Number in group	Number in group killed after 210 days	Number in group dying	Survival time in days
Controls on stock diet	4	4	0	[a]210+
Controls on artificial diet	[b]7	3	4	165+
A deficient	4	1	3	162+
D deficient	4	0	4	85
C deficient	4	0	4	95
B_2 complex deficient	4	0	4	76
B_2 complex controls	4	0	4	98
Nicotinic acid deficient	4	0	4	67
Flavin deficient	4	0	4	54

[a] The plus mark following days of survival means that one or more monkeys in the group were chloroformed in order to terminate the experiment, so it is impossible to calculate the complete survival time of the group.
[b] 1 animal died on the seventh day and was therefore excluded from consideration in this table.

Table 2 summarizes the survival time for the various groups, regardless of the cause of death. Since all of the monkeys were kept in individual cages in the two rooms, the effects of the various diets may have had some effect on their cage; age at death. If this is true, it

TABLE 3.—*Mouth lesions observed at death in monkeys on various diets tested*

(Condensation of data presented in table 1)

Group	Total number in group	Number in group affected and weighted severity of lesions	Gingivitis	Necrosis of interdental papillae	Necrotic gingivitis	Ulcerations of buccal mucosa	Noma
Controls on stock diet	4	Number in group affected.	0	0	0	0	0
		Weighted severity of lesions.	0	0	0	0	0
Controls on artificial diet	*7	Number in group affected.	1	1	0	0	0
		Weighted severity of lesions.	0.1	0.1	0	0	0
"A" deficient	4	Number in group affected.	1	1	0	0	0
		Weighted severity of lesions.	0.3	0.3	0	0	0
"D" deficient	4	Number in group affected.	4	2	2	0	0
		Weighted severity of lesions.	1.5	1.0	0.5	0	0
"C" deficient	4	Number in group affected.	4	4	3	0	0
		Weighted severity of lesions.	3.5	3.3	2.5	0	0
B_2 complex deficient+ supplements of flavin and nicotinic acid.	4	Number in group affected.	4	3	3	1	1
		Weighted severity of lesions.	3.8	1.8	2.3	1.0	1.0
B_2 complex deficient+ supplement of nicotinic acid.	4	Number in group affected.	2	2	2	1	1
		Weighted severity of lesions.	1.8	1.5	1.3	1.0	1.0
B_2 complex deficient+ supplement of flavin.	4	Number in group affected.	2	2	2	2	0
		Weighted severity of lesions.	1.8	1.8	1.5	1.3	0
B_2 complex deficient	4	Number in group affected.	4	4	3	2	1
		Weighted severity of lesions.	3.5	3.3	2.8	1.8	1.0

* 1 animal died seventh day; therefore excluded from consideration in this table.

perhaps was at play as a factor in host resistance to spontaneous infection. As striking as these results are, it must be remembered that the groups are small, and before much reliance can be placed on such a possible correlation many more experiments of this type must be made.

In table 3 the data on mouth lesions have been condensed. The less severe lesions (i. e., soft tartar, localized recession, and inflammation) were deleted so that differences between the groups might be more evident. In addition, in order to take into account both the number of animals affected and the severity of the lesions present in those animals involved, a process of "weighting" has been made. This has consisted of adding the numbers denoting degree of severity in each animal and dividing by the number of animals in the group. From this table it is apparent that both the control diets protected the monkeys from developing mouth lesions, and the diet supposedly deficient in vitamin A was about as efficient in this respect as the artificial control diet. In the remaining deficiency groups, however, there is a definite difference in the numbers developing lesions and the severity of these lesions. Again the point is stressed that only in

some part of the B_2 complex deficiency did lesions in the buccal mucosa develop, and in these cases only did noma appear.

ATTEMPTS AT TRANSMISSION OF ORGANISM FROM INFECTED LESIONS TO NORMAL MONKEYS ON A STOCK DIET

In order to test the infectivity and the virulence of the spirals, fusiforms, and other organisms occurring in the mouths of those monkeys developing lesions, it was decided to try several direct transfers of this material to the mouths of normal monkeys. The general plan was thoroughly to swab the lesions with a saline-soaked cotton swab, emulsify this immediately in about 3 cc. of saline, and then to use this suspension at once to inoculate the mouths of normal monkeys on stock diet 495a. Following this, the suspension was studied in stained smears and under the dark field to establish the fact that large numbers of viable (by motility) organisms were actually inoculated. The animals were all observed daily for evidence of infection. The individual protocols are presented in table 4, where it is noted that no gingivitis or stomatitis resulted from this procedure.

TABLE 4.—*Data on attempts at transmission*

Source of inoculum						Inoculated		
Date	Monkey No.	Deficiency	Description of mouth lesion on date of transfer	Spirals and fusiforms [1]	Motility	Transferred to monkey No.	Preparation of gums for transfer	Results
June 14, 1938	52	B_2 complex	Noma of right cheek. Beginning noma of left.	2+	Motile	451	Deep swabbing of socket of recently extracted superior central incisor.	Negative after 2 weeks.
						373	Laceration of buccal mucosa.	Do.
						345	Laceration of interdental papillae.	Do.
						70	No trauma	Do.
June 17, 1938	35	B_2 complex	Acute gingivitis with necrotic area upper central incisor area.	4+	Motile	452	No trauma	Negative after 2 weeks.
						453	Laceration of buccal mucosa.	Do.
						454	Laceration of interdental papillae.	Do.
June 17, 1938	40	"C"	Acute generalized gingivitis.	3+	Motile	455	No trauma	Negative after 2 weeks.
						456	Laceration of buccal mucosa.	Do.
						457	Laceration of interdental papillae.	Do.

[1] 2+=small number. 3+=moderate number. 4+=large number.

It would seem from these 3 separate attempts at transmission to 10 normal animals on a stock diet that even though spirals and fusi-

forms were present in relatively large numbers, they were incapable of initiating primary or secondary lesions regardless of whether the tissues had been previously traumatized. Furthermore, other organisms present in the saline suspension were also incapable of initiating lesions in the mouths of normal monkeys.

One monkey, 451, deserves an added comment. In this monkey the inoculation was made by a deep swab of the socket of a superior central incisor which had been removed several hours before. This should have furnished adequate anaerobiosis for the growth of the organisms, yet healing went on normally and at no time were any unusual reactions noted.

SUMMARY AND CONCLUSIONS

Although the number of animals is relatively small, observations on *Macacus rhesus* monkeys are presented which indicate that certain of the dietary deficiencies tested are associated with the development of varying manifestations of gingivitis, stomatitis, periodontal disease, and noma. Comparable animals maintained on adequate diets showed little or no evidence of such oral pathology. In addition, monkeys maintained on a stock diet showed no evidence of gingivitis or stomatitis when material from affected monkeys was transferred directly to them.

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ORAL PATHOLOGY IN MONKEYS IN VARIOUS EXPERIMENTAL DIETARY DEFICIENCIES *

By T. H. Tomlinson, Jr., *Passed Assistant Surgeon, United States Public Health Service*

INTRODUCTION

Topping and Fraser [1] have stated the objectives and outlined the methods of procedure followed in their experiment. This paper will

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[1] Topping, N. H., and Fraser, H. F.: Mouth lesions in monkeys associated with dietary deficiencies. Preceding article in this issue.

report the oral pathology found in the 39 *Macacus rhesus* monkeys included in their study.

No reports have been found in the literature giving the histopathology of oral lesions in monkeys from experiments conducted simultaneously relating to the influence of vitamins A, C, and D, and nicotinic acid and flavin.

MATERIAL AND METHODS

Since changes in the soft tissues were to be studied rather than those occurring primarily in the teeth or bone, ground sections were not made. The questions of bone resorption, osteoclasia, new bone formation, and tooth development will be omitted from this paper and may be reported later when additional studies with ground sections are made.

In securing the blocks for histopathological study, the entire head was split in the median sagittal plane and fixed in 10 percent formalin. The jaws were disarticulated and, with each half held firmly in a clamp, a small, fine to medium toothed hack-saw was used to take a transverse section about 5 mm. in thickness through the alveolar process from both the right and left sides of the mandible and maxilla. Some sections were taken through the erupted teeth and others through the interdental regions. When gross lesions were evident, the sections were taken at these points.

These blocks were decalcified in 22.5 percent formic acid in 10 percent sodium citrate solution. Each was placed in 30 cc. of this decalcifying solution for 1 week, at the end of which time the acid was poured off and fresh acid added for a period of another 7 to 10 days, depending on the hardness of the tissue at that time. It was found that routine decalcification for 3 weeks or longer resulted in poor cellular detail as regards infiltrating leucocytes, while less than 2 weeks' treatment left the section too hard for satisfactory sectioning. Sixteen to eighteen days appeared to give the most satisfactory results.

It was felt that minute cellular detail was a little better in this decalcified material when the paraffin sections were stained with hematoxylin-eosin than when the hematoxylin-Romanowsky technique was used. In addition, sections stained by Van Gieson's picrofuchsin were studied on each animal.

In recording findings the sections were divided into four parts—the free gingiva, the periodontal membrane, the intercancellous tissue, and bone. The free gingiva was subdivided into the tip, the lateral mucosa, the part lining and subjacent to the crevice, and the epithelial attachment with its subjacent fibrous tissue.

CONTROL ANIMALS

The changes found in the four animals on *stock diet No. 495a* consisted of slight to moderate lymphocyte and plasma cell infiltration of the corium of the free gingiva, usually beneath the crevice and the epithelial attachment, with occasional small foci of lymphocytes and neutrophils in the crevice epithelium. In 3 animals, 4 areas out of 16 showed slight thickening of the epithelial attachment accompanied in 1 area by a few rete pegs entering the corium.

In the 7 animals on *basic diet No. 482 with appropriate supplements to render it presumably adequate*, there was moderate to marked focally dense round cell infiltration of the corium of the free gingiva which generally involved the tip as well as the crevice and attachment regions. Animal No. 58 showed in two areas pericellular edema and neutrophil infiltration of the epithelium with loss of superficial cells from the tip to the cemento-enamel junction. Nos. 50 and 69 each showed ulceration in a similar location, while the latter also showed necrosis of the entire free gingiva on the labial side in one section. Monkey 39 showed marked focal perivascular round cell infiltration in the walls of the tooth follicles. This was not seen in any of the other 38 animals. The epithelial attachment showed in 3 monkeys occasional areas of necrosis and in 3 others slight to moderate thickening accompanied in 2 of them by numerous rete pegs.

Since most of the reported work on oral pathology as related to vitamin deficiencies had been carried out on guinea pigs, rats, and dogs, it was felt that considerable attention should be directed at the beginning of this work toward the determination of the possible significance in *Macacus rhesus* of round cell infiltration in the corium of the free gingiva. In addition to the 11 controls on this experiment, a few monkeys, most of which had been used in virus work, were examined, from 4 to 12 sections being taken from each. Occasional sections were obtained from almost all of these animals showing a thin, compact epithelial attachment with no rete pegs and no leucocytes in the corium of the free gingiva, or, at the most, a dozen or so scattered lymphocytes: However, about one-half or more of the sections from every monkey showed cellular infiltration as described above either in the lateral or medial free gingiva, occasionally in both.

In view of these findings it was not felt that much significance could be attached to round cell infiltration beneath the crevice and attachment epithelium when occurring alone, at least not in *Macacus rhesus* as fed, housed, and handled in our laboratory.

VITAMIN A DEFICIENCY [2]

Here there was no more than a very slight keratosis of the alveolar epithelium. There were occasional small foci of edema and lymphocyte and neutrophil infiltration in the epithelium of the crevice and attachment. In animal 51 the crevice and attachment epithelium was necrotic, and No. 46 showed ulceration and marked cellular infiltration of the epithelium of one interdental papilla. Beneath the areas of maximum epithelial involvement there were many round cells and often a few neutrophils. The plasma cell and lymphocyte infiltration of the corium of the free gingiva was moderate in two animals and marked in the others. There was slight to moderate thickening of the epithelial attachment in three animals accompanied in two of them by a few rete pegs.

D DEFICIENCY

Three out of the four monkeys showed in one-fourth to one-half of the sections taken from each animal complete necrosis of the free gingiva, the term complete necrosis meaning loss of cellular detail, usually with, but occasionally without, preservation of stroma outline. In one of these there was also moderately extensive necrosis of the periodontal membrane and intercancellous tissue and some focal bone necrosis. This monkey also showed hemorrhage and much free and phagocytosed blood pigment in the areas of necrosis and infiltration. In the other two there was moderate to marked focal lymphocyte and neutrophil infiltration of the intercancellous tissue. In the gingiva of the fourth monkey (No. 53) and in the surviving gingiva of the others there were occasional foci of neutrophils and usually moderate, often dense, lymphocyte and plasma cell infiltration of the corium often extending from the tip to the cementoenamel junction as a wide zone beneath the epithelium. This animal also showed slight thickening of the epithelial attachment with a few rete pegs.

C DEFICIENCY

In this group all four animals showed extensive necrosis of the gingiva with, in one of them, moderately extensive necrosis of the periodontal membrane, intercancellous tissue, and bone. In the surviving areas there was marked neutrophil, lymphocyte, and plasma cell infiltration of the gingiva occasionally extending into the periodontal membrane and the intercancellous tissue. The areas of gingival involvement often showed occasional to numerous, small to large hemorrhages, much free and phagocytosed golden brown granu-

[2] That the depletion of vitamin A in this group may have been a relative one is pointed out by Topping and Fraser in their paper.

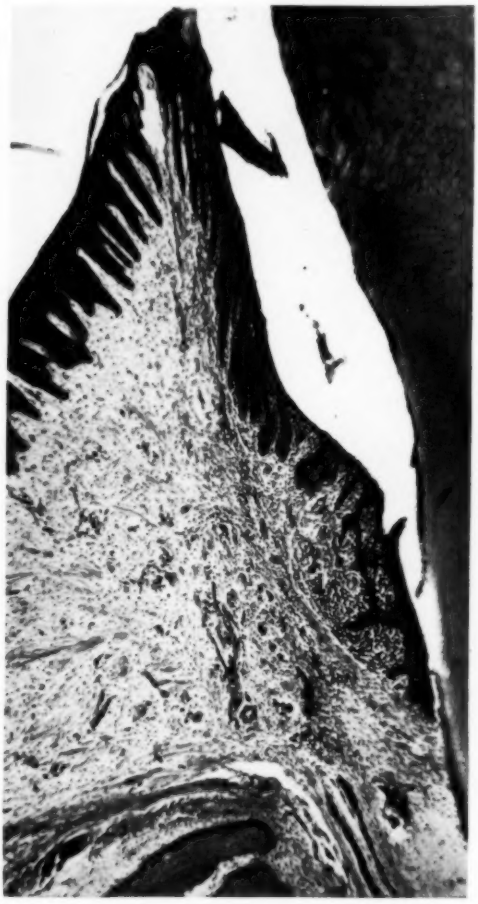

FIGURE 1.—No. 61. Stock diet 495a. Thickening of epithelial attachment with rete pegs. Round cell infiltration beneath attachment and bottom of crevice. × 42. (N. I. H. 994.)

FIGURE 2.—No. 40. C deficiency. Necrosis and focal suppuration in free gingiva with subjacent plasma cell and lymphocyte infiltration, fibroblast proliferation, little free blood, and free and phagocytosed blood pigment. × 42. (N. I. H. 992.)

FIGURE 3.—No. 43. D deficiency. Necrosis of free gingiva. Subjacent round cell and neutrophil infiltration and hemorrhage extending into the intercancellous tissue, peridental membrane, medial alveolar mucosa, and periosteum of medial alveolar bone plate. Focal necrosis of alveolar bone crest. Much blood pigment. × 16. (N. I. H. 1000.)

FIGURE 4.—No. 72. Nicotinic acid deficiency. Necrosis of free gingiva with subjacent round cell and neutrophil infiltration. Focal hemorrhage above alveolar bone crest on labial side. × 16. (N. I. H. 1004.)

FIGURE 5.—No. 30. B_2 complex deficiency plus nicotinic acid and flavin. Extensive labial gingival necrosis extending into peridental membrane. Hemorrhage in gingiva and peridental membrane. × 16. (N. I. H. 989.)

lar pigment giving the prussion blue reaction, and some darker iron-free pigment. This pigment was more abundant in the free gingiva, but there were scattered and clumped pigmented macrophages in the corium of the lateral alveolar mucosa some distance down from the alveolar crest. A little free and phagocytosed blood pigment with occasional small hemorrhages were seen in the intercancellous tissue.

B_2 COMPLEX DEFICIENCY PLUS NICOTINIC ACID AND FLAVIN [3]

In this group one monkey showed slight focal superficial necrosis of an interdental papilla and in the other three there was variably extensive necrosis in all parts of the gingiva. In No. 30 the necrosis was focally suppurative but more often hemorrhagic and involved to a moderate degree the periodontal membrane. There was also extensive hemorrhage and much blood pigment in the gingiva and periodontal membrane. Monkey No. 63 showed a large perforating lesion of the left cheek, a section through the margin of which showed complete necrosis of all tissues with little marginating inflammatory reaction. In a cross section of the upper jaw beneath this lesion there was complete necrosis involving the internal free gingiva, the external gingiva, the maxillary mucosa, and the bucco-maxillary sulcus. The periodontal membrane, the intercancellous tissue in areas, and the bone throughout most of the section were necrotic.

FLAVIN DEFICIENCY

Monkey No. 52 showed an area of necrosis in the right cheek 32 mm. in diameter and a similar-8 mm. area on the left side, both centrally perforated. Marginating the necrotic areas were diffuse round cell and neutrophil infiltration and moderate edema. Beneath both of these areas the upper jaws showed complete necrosis of the gingiva and of the intercancellous and peritrabecular tissues, the periodontal membrane, and most of the bone throughout the distal half of the alveolar process, including often the lateral and medial alveolar plates. In one section there was an old hemorrhage in the region of the tooth socket. The other three animals showed usually slight to moderate, sometimes focal, lymphocyte and plasma cell infiltration of the corium, usually beneath the crevice epithelium and the epithelial attachment, less frequently in the tip of the free gingiva. Two of these three showed very slight focal superficial necrosis of the gingiva and the other one showed a moderately thickened epithelial attachment with numerous rete pegs.

[3] The adequacy of this diet as a B_2 complex control for monkeys was discussed by Topping and Fraser in their paper. The extensive pathology found in these animals suggests that one or more other deficiency factors may be involved.

NICOTINIC ACID DEFICIENCY

In this group No. 38 and No. 72 showed extensive, usually complete, occasionally suppurative necrosis of the free gingiva involving also the lateral alveolar mucosa. The subjacent surviving soft tissues showed round cell and neutrophil infiltration. Both monkeys showed focal extension of the infiltration and the necrosis into the periodontal membrane and the intercancellous tissue, with slight marginal necrosis of adjacent bone in No. 38. Monkey No. 72 showed on the inner surface of the lower lip a small circumscribed area of complete necrosis of the epithelium and subjacent corium, marginated by lymphocyte and neutrophil infiltration. The other two animals in this group showed slight to moderate, often focal round cell infiltration of the corium of the free gingiva, with moderate thickening of the epithelial attachment.

FLAVIN AND NICOTINIC ACID DEFICIENCY

One monkey (No. 77) from this group showed in one section ulceration of the tip of the free gingiva. The free gingiva of the other three showed extensive suppuration and necrosis, restricted in one animal (No. 62) to the labial free gingiva but involving the gingiva of the entire alveolar process in the other two and extending in areas below the alveolar bone crest. In these two (Nos. 35 and 64) there was moderately extensive necrosis of the periodontal membrane, and in No. 35 the intercancellous tissue, in areas throughout the distal half of the section, was necrotic and surrounded a few fragments of dead bone. Nos. 62 and 77 showed moderate to marked round cell infiltration of the corium of the surviving gingiva with many rete pegs entering from the attachment. Monkey 35 showed on the oral surface of the upper lip a 1 cm. penetrating ulcer and on the lower lip a similar 3½ by 2 cm. lesion extending to the mucocutaneous border. Sections from these areas showed complete necrosis of the oral mucosa extending about one-half way through the subjacent fibrous tissue and muscle except at the crest of the lips, where it extended completely through and involved the epidermis. Marginating inflammatory reaction was very slight. Animal No. 62 showed ulceration of the buccal mucosa opposite the areas of gingival necrosis.

DISCUSSION

The epithelial attachment showed some alteration in most of the animals, varying from slight focal thickening to complete necrosis; however, six monkeys showed no changes in any of the sections examined. Types of involvement and group distribution are shown in table 1.

TABLE 1.—*Epithelial attachment* [1]

Diet and number of animals	Normal	Thickening	Rete pegs	Necrosis
Stock 495a (control): (4)	65	34∓ 60± 61+	61+	
482 plus supplements (control): (7)	55 79	39++ 41± 50++	39++ 50++	50± 58∓ 69+
A deficient: (4)	68	36∓ 46+ 51++	46+ 51+	51+
D deficient: (4)		53+	53+	43+++ 44++ 75+++
C deficient: (4)				40+++ 47± 57+++ 71+++
B_2 complex deficient plus nicotinic acid and flavin: (4)	48	63++		30+++ 63++ 56+
Flavin deficient: (4)	78	45++	45++	49+ 52+++
Nicotinic acid deficient: (4)		38++ 67++	59±	38+++ 72+++
Flavin and nicotinic acid deficient: (4)		77++	62++ 77++	35+++ 62++ 64+++ 77+

[1] Figures are animal identification numbers and + and − signs indicate degree of pathological condition.

Since all animals on this experiment showed cellular infiltration of the free gingiva, no attempt is made to evaluate this finding. There was round cell infiltration of the intercancellous tissue without necrosis in three monkeys, and perivascular round cell infiltration in the walls of the tooth follicles in one.

Blood pigment or hemorrhage was present, and occasionally both, in sections from all of the C deficiency animals, one of the D deficiency, one of the flavin deficiency, and one of the group on B_2 complex deficiency plus flavin and nicotinic acid. Sections showing brown pigment in the tissues were stained for hemosiderin by the Perls-Abbott method, sometimes with negative results, but frequently a positive prussian blue reaction was obtained. This variability was expected in accord with Lillie's [4] recent studies on the solubility of hemosiderin in acids.

Necrosis (table 2) was not seen in any of the animals on stock diet 495a and was present in the gingiva of only 3 on diet 482 plus appropriate supplements. Among the remaining 28 monkeys, 16 showed

[4] Lillie, R. D.: Experiments on the solubility of hemosiderin in acids and other reagents during and after various fixations. In press.

extensive gingival necrosis and 6 others showed ulceration, either of the free gingiva or the interdental papillae. There was slight to extensive necrosis of the periodontal membrane and intercancellous tissue in 7 of these and of the periodontal membrane alone in 2 others. Necrosis of bone was seen in 6 animals and always in association with inflammation and necrosis of adjacent soft tissues. The necrosis of the gingiva and related structures was fairly evenly distributed throughout the various deficiencies with the exception of the (partial?) A deficiency group where the animals showed only slight focal necrosis of the gingiva. In all of the other groups from 2 to 4 animals showed rather extensive gingival necrosis with 1 to 2 monkeys from each group showing in addition necrosis of the periodontal membrane, intercancellous tissue, and bone.

TABLE 2.—*Necrosis* [1]

Diet and number of animals	Gingiva	Periodontal membrane	Intercancellous tissue	Bone	Cheeks and lips
Stock 495a (control): (4)					
482 plus supplements (control): (7)	58∓ 50± 69++				
A deficient: (4)	46± 51+				
D deficient: (4)	43+++ 44++ 75+++	43++	43++	43+	
C deficient: (4)	40+++ 47+++ 57+++ 71+++	40++	40++	40++	
B_2 complex deficient plus flavin and nicotinic acid: (4)	30+++ 48± 56++ 63+++	30++ 63+++	63+++	63+++	63 (Noma)
Flavin deficient: (4)	49± 52+++ 78±	52+++	52+++	52+++	52 (Noma)
Nicotinic acid deficient: (4)	38+++ 72+++	38+ 72++	38+ 72+	38+	72
Flavin and nicotinic acid deficient: (4)	35+++ 62+++ 64+++ 77±	35++ 64++	35+++	35++	35 (Noma) 62

[1] Figures are animal identification numbers and + and − signs indicate degree of pathological condition.

Among the 16 monkeys fed a B_2 complex deficient diet with variations only in the flavin and nicotinic acid factors, 1 showed marked ulceration of the buccal mucosa, 1 a large circumscribed area of necrosis on the oral surface of the lower lip, and 3 showed perforating necrotic lesions of the cheeks or lips.

SUMMARY

The feeding of 39 monkeys on diets deficient in certain vitamins resulted in the development of necrosis of the periodontal tissues and

oral mucosa of varying extent, qualitatively similar in the B_2 complex deficiencies with variations in the flavin and nicotinic acid factors, in the C deficiency, and in the D deficiency, and probably greatest in extent in the B_2 deficient groups.

A STUDY OF EXPERIMENTAL PERTUSSIS IN THE YOUNG RAT*

By J. W. HORNIBROOK and L. L. ASHBURN, *Passed Assistant Surgeons, United States Public Health Service*

Burnet and Timmins (*1*) showed that the intranasal instillation of *Haemophilus pertussis* in the mouse produced pneumonia and death. This has been confirmed by Bradford (*2*) and Culotta (*3*). One of us found that young rats more uniformly developed a fatal pneumonic infection than did mice when inoculated in this manner, and that the required infecting dose was considerably smaller for rats than for mice. Thus approximately 8,500,000 phase I organisms cause infection in the rat as compared with 200,000,000 in the mouse. Those rats which do not die early of pneumonia usually develop paroxysms in which air is forcibly expelled from the mouth or nose. This resembles a cough or sneeze. Though adult rats seldom die from the inoculation, they develop a more vigorous cough than the younger animals. The cough can be heard a distance of 10 to 20 feet. Vomiting was not observed.

INFECTION IN THE RAT

Method of inoculation.—A loopful of a 24-hour growth of phase I *H. pertussis* from Bordet Gengou media was suspended in saline. Using Burroughs Wellcome opacity tubes and the *H. influenzae* opacity table as a standard, an estimate was made of the number of organisms per cc. This suspension was then diluted so as to give an estimated 170,000,000 organisms per cc. The young rat (25–40 grams) was anesthetized with ether and held on its back, and the suspension was dropped into the nose with the aid of a syringe and blunt needle, the material being drawn into the respiratory tract during inspiration. The capacity of the lungs of young rats of the size mentioned rarely permits the instillation of volumes greater than 0.1 cc. The practice was to instill 0.05 cc. into each rat. If rhonchi were not felt, the rat was reanesthetized and the dose repeated.

Both Wistar albino and hooded rats were used in these experiments.

Results of inoculation.—In all, 51 animals were inoculated. Six were killed on the 2nd day and five on the 4th day for pathological examination. Twenty-six of the remaining 40 animals died on or

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before the 9th day, a mortality of 65 percent. The surviving animals were killed on or after the 10th day for section. Those animals dying of the disease survived an average of 5.2 days, with 9 days as the longest survival time and 3 days as the shortest. None of the rats showed any evidence of toxicity until a few hours before death. Animals which appeared perfectly well, except possibly for an increased respiratory rate, showed rather extensive lung changes at autopsy.

With a given strain of *H. pertussis* comparable results were obtained in each series of rats inoculated. On the other hand, when mice were used as the test animal the results were very irregular. A dose which killed in one series would often fail in another, though the technique and number of organisms used were the same.

A record of coughing was kept on 12 rats. The cough appeared in an average of 8.3 days following inoculation. The termination of the cough was difficult to estimate, as it disappeared so gradually. In some cases cough continued for 60 days.

Isolation of H. pertussis from the infected rat.—The lungs of 35 rats were cultured, with the following results:

Time inoculation to death, in days	Number of animals	Number of animals positive for pertussis
1–5	6	3
6–10	16	9
11–20	1	1
21–30	5	0
31–40	3	0
60	4	2

H. pertussis cultures were recovered from only 43 percent of the 35 rats. This low percentage may be explained in part by the fact that many cultures were overgrown with spreading colonies so as to obscure any *H. pertussis* which might have been present. The tabulated results also include earlier work when cultures were made by means of tracheal swabs. Subsequently one lung from each animal was ground and used as the inoculum. This produced a higher percent of positive cultures.

The organism isolated from the 60-day animals seemed to grow more rapidly than usual. With phase I antiserum this organism agglutinated in a dilution of 1:540, while fresh phase I bacilli agglutinated in a dilution of 1:1620 with the same serum.

Pathology.—The absence of post mortem changes made 31 animals suitable for pathological study; and of these, all but five showed microscopic evidence of lung changes. As is to be expected, the microscopic findings in these rats showed progressive changes with prolongation of the infection.

The rat lung reacts to the introduction of *H. pertussis* by a process generally described as interstitial bronchopneumonia. In the early stages the exudate in the alveoli is by far the most striking feature; later, as this is resolved, the interstitial reaction becomes prominent. The number of consolidated areas in one lobe, the size of these areas, and the number of lobes involved are quite variable; also, the separate foci frequently have their inception at varying intervals, and consequently many stages of the developing process may be seen in the same lobe or different lobes of the two lungs.

The earliest reaction was seen about 48 hours following inoculation. There is slight congestion of the alveolar capillaries and slight to moderate serum exudation into alveoli. The areas involved are generally small, are in a peri- or para-bronchial location, and usually near the hilus. Not all alveoli in a given area are involved; some are solidly filled, although most show incomplete filling, and few have only a thin coating on the surface of septa. Interspersed with these are varying numbers of air-containing and occasionally dilated alveoli. In few scattered, and occasionally grouped, serum-containing alveoli there are occasional to few neutrophils, occasional small macrophages, and rarely a little fibrin. Alveolar septa show very slight neutrophil infiltration, with slight proliferation and enlargement of septal cells, few of which appear as macrophages. Small vessels show narrow surrounding zones of adventitial edema, occasionally with very few infiltrating neutrophils. The small bronchi show similar infiltration but without edema. Rarely a neutrophil is seen in the epithelium of small bronchi and bronchioles. A few small Gram-negative bacilli are scattered throughout alveolar serum and rarely in small groups on the surface of bronchial epithelium.

By the third day areas of involvement have become much larger, reaching nearly to the pleural surface, and occasionally an entire lobe is consolidated. Edema fluid is quite dense, and fewer alveoli contain air. Neutrophils in the exudate are much more numerous and occasionally show pyknosis and karyorrhexis; in some alveoli they are rather densely packed; generally they occur in moderate numbers; however, numerous scattered and grouped alveoli contain only serum, even in the older central area of the lesion. This irregular distribution of cells is also striking in more advanced stages. A few small and large macrophages are admixed with the neutrophils, but they are slightly more prominent in the less cellular exudate. Occasionally their ample cytoplasm contains oxyphilic hyalin globules. The septa show moderate irregular thickening, congestion, variable though usually moderate proliferation and swelling of septal cells, with few scattered neutrophils and fairly numerous macrophages and large mononuclear cells. These latter cells often protrude as knob-like projec-

tions from septa. The large vessels near the hilus show moderate perivascular serum exudation with moderate infiltration by neutrophils and few lymphocytes, mast cells, and macrophages. A similar but less prominent reaction is present around some smaller vessels. Around bronchi very small amounts of serum occur with occasional to few neutrophils. The epithelium of small bronchi and bronchioles is about twice its normal thickness, cells are considerably enlarged, particularly in height, and nuclei are oval, or round, and leptochromatic. The cilia-bearing surface is frayed and quite uneven. Little serum, few neutrophils, and occasional red cells are present in the lumen. Infrequently the lumen is completely filled with serum. At this stage bacteria are present in very great numbers, in clumps, and scattered throughout the alveolar exudate, and less numerous in bronchial exudate and on the surface of the epithelium. Few are phagocytosed.

From the third to fourth day there is simply an accentuation of the changes already established. More neutrophils show degenerative changes, serum is thinner and thready, macrophages in alveoli are more numerous and larger, and few to many binucleate or occasionally multinucleate cells are present. These latter cells have little basophilic cytoplasm and their large nuclei are deeply stained and vesicular. Septa show slightly greater thickening and in many areas are lined by discrete or coherent, large, occasionally vacuolated epithelial cells, few of which lie free in the alveoli. Occasionally septal cell proliferation and swelling of epithelial cells completely obliterate alveolar spaces. The perivascular and peribronchial reaction shows little progression. Cells are slightly more numerous, with relative and actual increase in mononuclear cells, mainly macrophages. The fibroblasts are swollen but show no significant proliferation. Edema fluid is no longer seen in the adventitia of smaller vessels. Bronchial exudate and epithelium show no additional changes except a little focal serum exudation between epithelium and supporting tissues.

By the fifth to sixth day the alveolar exudate shows much necrosis and is decreased in amount; however, macrophages are still moderately numerous and usually of the large foamy type. Septa show no significant additional change. The striking feature is the increased prominence of the peribronchial and perivascular reaction, particularly near the hilus. In addition to considerable increase in number of cells, there is moderate fibroblast proliferation. The infiltration and proliferation occupy a wider zone than that previously seen and extend to involve the loose tissue about the lung roots, and occasionally extend along the pulmonary vessels to the base of heart. Perivascular and peribronchial edema is no longer present. Epithelial cells of small bronchi and bronchioles still show hypertrophy and marginal fraying, and cells of larger bronchi are quite tall. In an

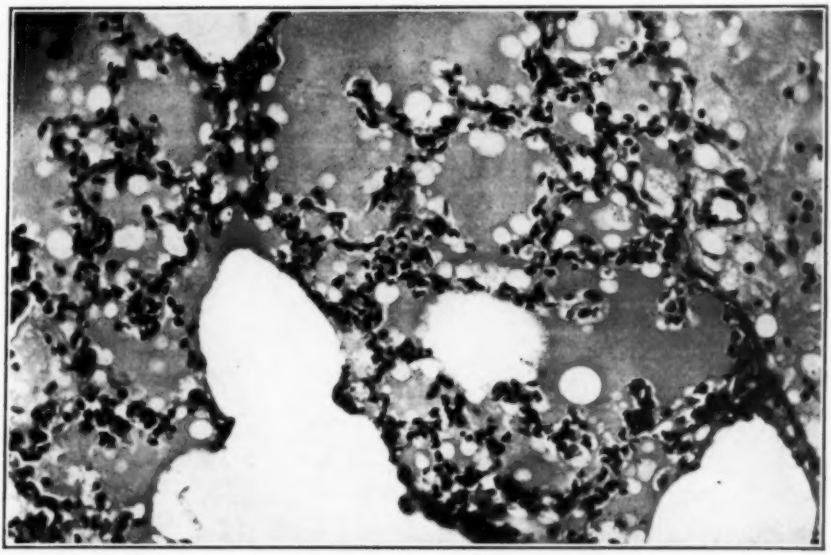

FIGURE 1.—Rat lung 2 days following intranasal inoculation with *H. pertussis* showing alveoli filled with serum and few emphysematous air spaces. Edema fluid seen in upper right part of photomicrograph is perivascular in location. × 250. (N. I. H. 1084.)

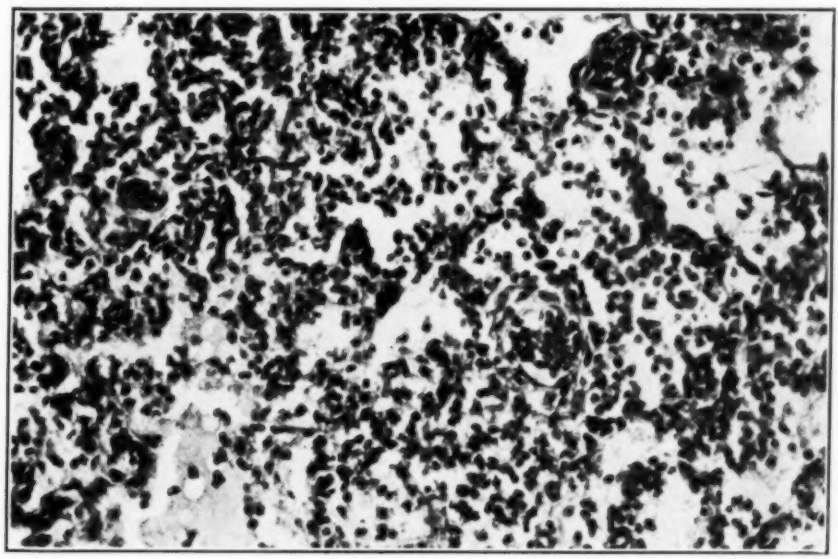

FIGURE 2.—Three-day lung lesion showing cellular exudate in alveoli, mainly neutrophils, also a little serum and fibrin. × 250. (N. I. H. 1052.)

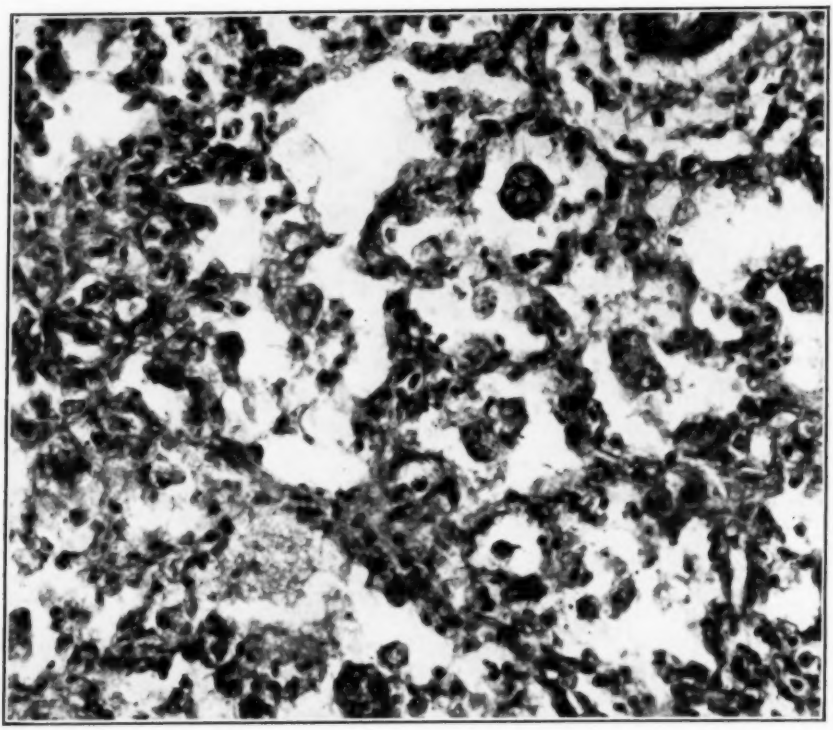

FIGURE 3.—Five-day lung lesion showing thickening of alveolar septa, increase in number and size of septal cells, and large mononuclear and multinuclear cells in alveoli. × 340. (N. I. H. 1051.)

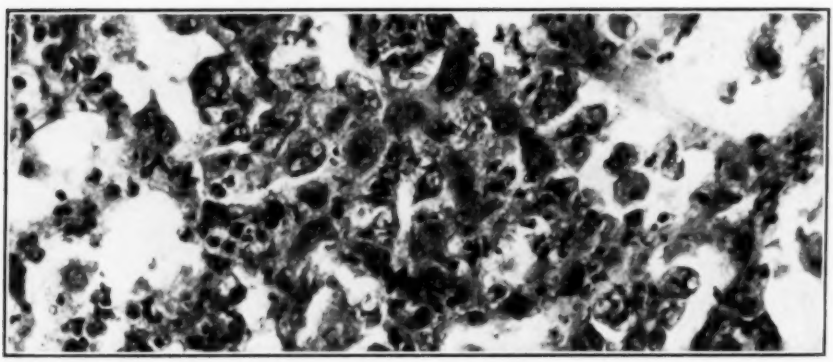

FIGURE 4.—Five-day lung lesion showing area in which some alveoli are obliterated by proliferation and exudation of large mononuclear and multinuclear cells. × 340. (N. I. H. 1053.)

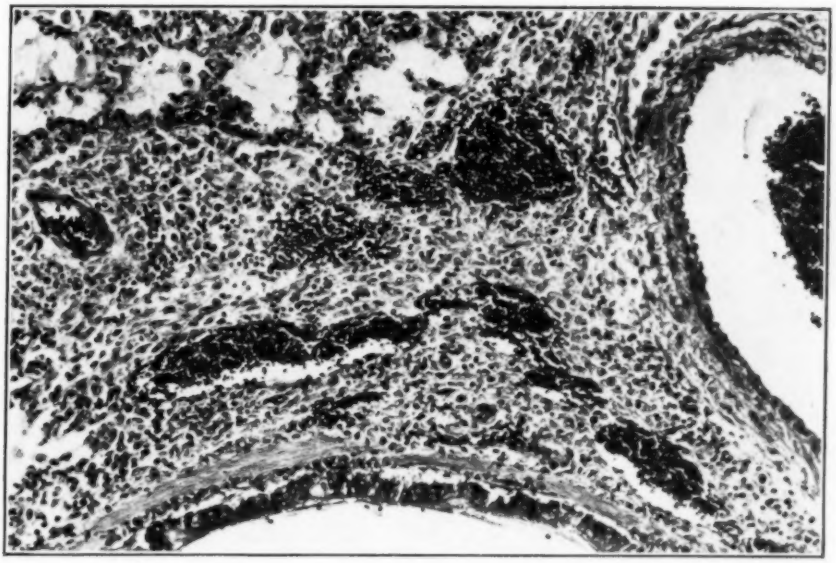

FIGURE 5.—Six-day lesion showing marked peribronchial and perivascular mononuclear cell infiltration and fibroblast proliferation. × 125. (N. I. H. 1058.)

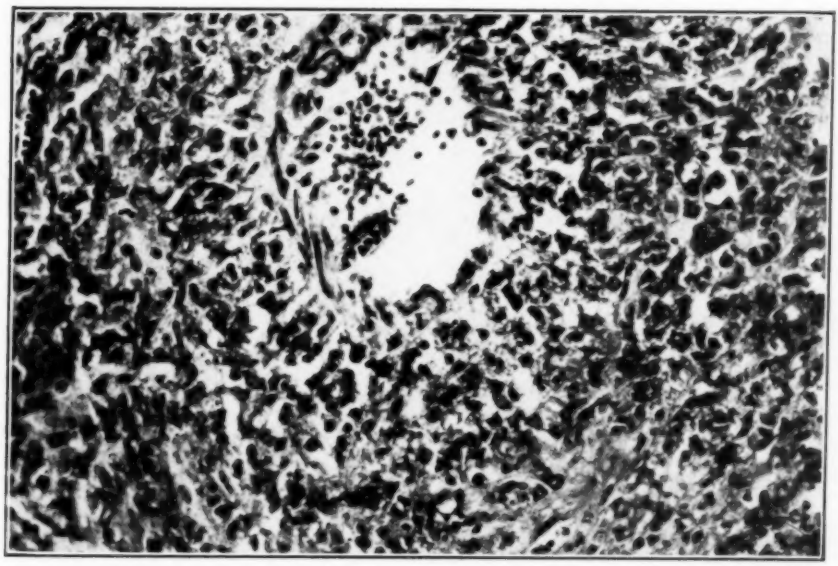

FIGURE 6.—Nine-day lung lesion. High-power photomicrograph of perivascular reaction, similar to that seen in figure 5. × 250. (N. I. H. 1059.)

occasional animal a few epithelial cells of large bronchi contain mucus, and a few strands of mucus and a few neutrophils are inconstantly present adherent to the epithelial surface.

Usually by the tenth day certain areas show almost complete absorption of alveolar exudate with the exception of occasional to few remaining macrophages. Alveolar septa show less cell infiltration, and their increased thickness is due mainly to enlargement of cells lining alveoli, the alveoli being reduced in size. In this stage there is still slight to moderate peribronchial and perivascular fibroblast proliferation with slight infiltration by large mononuclear cells. Bronchial epithelium shows no significant alteration, although an occasional group of small Gram-negative bacilli occur among the cilia.

In an occasional animal unresolved pneumonic foci have been found as late as fifteen days. This may be due to the fact that these lesions have had their onset some time after inoculation. This factor makes it difficult to follow the chronological development of the lesions after the tenth day. Focal, peribronchial septal thickening with atelectasis was present in one animal 21 days after inoculation.

It seems that in animals that do not die of the pneumonia the lungs return to their normal appearance, except in an occasional case where one or more small abscesses occur. These were present in two animals killed on the tenth post-inoculation day and showed an incomplete peripheral layer of loosely disposed fibroblasts surrounding an area of suppuration in which occasional non-necrotic alveolar septa persist. Bacteria, usually in clumps and morphologically similar to those seen in earlier lesions were present in the necrotic exudate. No abscesses were seen in later stages, but it is evident that such a lesion would leave a residual scar.

The tracheal reaction is generally very slight and often absent. It consists of slight congestion of mucosal corium, with very few scattered neutrophils. Epithelium is not altered; occasionally, however, a clump of small gram negative bacilli was present among the cilia. Serum was rarely present in lumen.

Other organs examined, including heart, liver, kidney, thymus, and spleen, showed no significant alteration.

DISCUSSION

The lung reaction occurring in rats is similar to that described in mice, and as has been pointed out by Bradford (*2*) and others, is not specific for *H. pertussis*. An essentially similar lesion has been produced experimentally by bacterial toxins, viruses, and a variety of bacteria—*B. typhosus* (*E. typhosa*), *B. bronchisepticus*, *B. lepisepticum*, and *H. influenzae*.

In mice, Burnet and Timmins (*1*) found goblet cells in the bronchiolar epithelium, with a film of mucus on the surface, and Bradford

(*2*) noted considerable amounts of mucus and cellular exudate accumulated in alveolar spaces and much mucus on the surface of bronchial epithelium. However, Culotta and associates (*3*), also working with mice, make no mention of the occurrence of mucus but record the presence of "homogeneous pink staining material" in the lumina of bronchioles. This absence of mucus and the presence of serum as found by Culotta and associates is in agreement with the findings in rats. Since the stagnation of mucus is usually stated to be the cause of spasmodic cough in human pertussis, the absence of mucus secretion in most rats (which also appear to cough) is interesting. Also, mechanical blockage of cilia, as suggested by Mallory and Hornor (*4*), cannot account for the cough in rats since so few bacteria were present among cilia.

SUMMARY AND CONCLUSIONS

The intranasal instillation of live phase I *H. pertussis* organisms into young rats produces a nonspecific interstitial bronchopneumonia which often leads to death (65 percent in our series).

Phase I *H. pertussis* organisms can be recovered from the involved lungs, particularly if the entire lung is ground and used as the inoculum.

The young rat is superior to the mouse as an experimental animal because of the fewer organisms required to infect and because of the higher and more uniform death rate. Furthermore, the rat so infected usually develops cough-like paroxysms, a phenomenon which in itself has experimental significance.

REFERENCES

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(*3*) Culotta, C. S., Marting, F. L., and Liebow, A. A.: Whooping cough: Observations on experimental infection in mice and on attempts at active immunizations in mice and in ferrets. Yale J. Biol. and Med., **10**: 233–240 (1938).

(*4*) Mallory, F. B., and Hornor, A. A.: Pertussis: The histological lesion in the respiratory tract. J. Med. Res., **27**: 115 (November 1912).

INFLUENZA PREVALENCE

The week ended March 11, 1939, recorded another increase in the number of cases of influenza—18,135 cases being reported as compared with 14,288 for the preceding week.

The greatest prevalence is still confined principally to the four groups of Central States and the South Atlantic region, with a slight increase shown in the Mountain and Pacific States, especially New Mexico and Oregon. Considerable increases are noted during the current week, as compared with the preceding week, for Wisconsin (from 584 cases to 1,516), North Dakota (364 to 741), Virginia (1,509 to 1,991), North Carolina (97 to 386), Georgia (140 to 420), Kentucky (1,348 to 1,792), Tennessee (146 to 469), and Alabama (599 to 1,126). The New England and Middle Atlantic States remained comparatively free from the disease.

The effect of the mild epidemic prevalence of influenza is now reflected in the weekly reports from cities, which show an increase in the number of cases reported and in the number of deaths attributed to the disease, but it is not yet indicated by excess deaths from pneumonia. For the week ended March 4, 1939, a group of large cities scattered throughout the United States and having an aggregate population of approximately 33,000,000, reported 1,271 cases of influenza, with 190 deaths, as compared with 5-year averages of 736 and 128, respectively; these same cities reported 891 deaths from pneumonia as compared with a 5-year average of 989.

The seasonal peak of influenza will evidently come much later this year than in prior years. In the past 11 years there have been three more or less mild influenza epidemics—1928–29, 1932–33, and 1937. In the first two the rise was rapid in November and December, the earlier epidemic reaching the peak of incidence in January, while the 1932–33 outbreak reached its peak in December. In 1937 the sharp rise occurred early in January and the peak was reached during the week of January 30, when 37,101 cases were reported.

The accompanying table gives the numbers of cases of influenza reported by States, arranged by geographical divisions and by weeks, from the first of the year to and including the week ended March 11.

126359°—39——3

Cases of influenza reported by weeks, Jan. 1–Mar. 11, 1939

Division and State	Jan. 7	Jan. 14	Jan. 21	Jan. 28	Feb. 4	Feb. 11	Feb. 18	Feb. 25	Mar. 4	Mar. 11
NEW ENGLAND										
Maine	1	3	2	10	4	1	8	25	46	103
New Hampshire				1						
Vermont										
Massachusetts										
Rhode Island										1
Connecticut	10	6	13	4	7	26	22	29	30	141
MIDDLE ATLANTIC										
New York	44	57	37	155	159	183	137	101	91	57
New Jersey	14	24	12	19	56	61	99	44	24	19
Pennsylvania										
EAST NORTH CENTRAL										
Ohio										
Indiana	12	11	22	4	21	21	363	1,085	607	35
Illinois	18	12	60	30	36	227	955	1,478	1,241	838
Michigan			1	2		1	39	255	429	674
Wisconsin	62	65	52	47	68	65	56	346	584	1,516
WEST NORTH CENTRAL										
Minnesota		2	3	2		1	3	24	12	40
Iowa		4	10	2	1	8	27	291	1,083	695
Missouri	70	59	24	33	24	42	137		644	678
North Dakota	34	11	12	6	27	15	14	64	364	741
South Dakota	6			2	1	10	3	6	77	50
Nebraska				1					2	1
Kansas	16	9	9	6	6	3	9	77	116	226
SOUTH ATLANTIC										
Delaware										
Maryland	4	5	12	10	61	103	182	209	124	53
District of Columbia	2	2	6		5	5	18	25	25	11
Virginia	454	420	282	617	1,100	553	1,338	1,604	1,509	1,991
West Virginia	21	13	34	41	21	26	33	36	271	71
North Carolina	3	7	28	9	9	18	71	230	97	386
South Carolina	909	495	865	649	772	701	972	592	1,181	1,142
Georgia	133	136	143	110	131	118	139	110	140	420
Florida	1	1	2	5		1	1		9	3
EAST SOUTH CENTRAL										
Kentucky	56	65	37	27	198	51	478	405	1,348	1,792
Tennessee	36	64	87	109	58	75	63	83	146	469
Alabama	158	191	188	169	259	186	160	180	599	1,126
Mississippi										
WEST SOUTH CENTRAL										
Arkansas	181	203	145	139	159	87	113	182	1,473	1,532
Louisiana	7	36	12	8	10	20	11	9	30	82
Oklahoma	222	149	119	193	162	207	129	193	334	387
Texas	492	716	531	703	699	621	983	737	965	968
MOUNTAIN										
Montana	5	26	33	50	25	42	35	200	126	125
Idaho	4	2	1	1	1			12	1	14
Wyoming									1	8
Colorado	21	21	31	45	35	93	125	121	150	136
New Mexico	2	1	21	10	6	9	1	3	57	677
Arizona	138	117	132	81	68	114	82	94	144	191
Utah	7	1	2	9	20	24	16	44	53	119
PACIFIC										
Washington		4	1			1	3		8	3
Oregon	71	39	46	53	25	40	42	34	97	261
California	41	41	82	33	76	43	28	59	50	73
Total	3,255	3,018	3,097	3,395	4,310	3,602	6,895	8,987	14,288	18,135

DEATHS DURING WEEK ENDED FEBRUARY 25, 1939

[From the Weekly Health Index, issued by the Bureau of the Census, Department of Commerce]

	Week ended Feb. 25, 1939	Corresponding week, 1938
Data from 88 large cities of the United States:		
Total deaths	10,086	[1] 8,608
Average for 3 prior years	[1] 9,713	
Total deaths, first 8 weeks of year	75,530	72,099
Deaths under 1 year of age	589	[1] 554
Average for 3 prior years	[1] 594	
Deaths under 1 year of age, first 8 weeks of year	4,433	4,332
Data from industrial insurance companies:		
Policies in force	68,013,875	69,772,226
Number of death claims	12,447	12,091
Death claims per 1,000 policies in force, annual rate	9.5	9.0
Death claims per 1,000 policies, first 8 weeks of year, annual rate	10.0	10.0

[1] Data for 86 cities.

PREVALENCE OF DISEASE

No health department, State or local, can effectively prevent or control disease without knowledge of when, where, and under what conditions cases are occurring

UNITED STATES

CURRENT WEEKLY STATE REPORTS

These reports are preliminary, and the figures are subject to change when later returns are received by the State health officers.

In these and the following tables, a zero (0) indicates a positive report and has the same significance as any other figure, while leaders (----) represent no report, with the implication that cases or deaths may have occurred but were not reported to the State health officer.

Cases of certain diseases reported by telegraph by State health officers for the week ended March 4, 1939, rates per 100,000 population (annual basis), and comparison with corresponding week of 1938 and 5-year median

Division and State	Diphtheria				Influenza				Measles			
	Mar. 4, 1939, rate	Mar. 4, 1939, cases	Mar. 5, 1938, cases	1934–38, median	Mar. 4, 1939, rate	Mar. 4, 1939, cases	Mar. 5, 1938, cases	1934–38, median	Mar. 4, 1939, rate	Mar. 4, 1939, cases	Mar. 5, 1938, cases	1934–38, median
NEW ENG.												
Maine	30	6	8	0	278	46	3	6	60	10	165	165
New Hampshire	0	0	0	0	----	----	----	----	91	9	33	30
Vermont	0	0	2	1	----	----	----	----	308	23	172	44
Massachusetts	6	5	5	8	----	----	----	----	1,248	1,061	242	916
Rhode Island	0	0	1	1	----	----	----	----	107	14	1	43
Connecticut	0	0	8	4	89	30	2	21	1,454	490	8	91
MID. ATL.												
New York	9	23	31	42	[1] 63	[1] 91	[1] 18	[1] 32	490	1,224	1,848	1,848
New Jersey	12	10	14	16	29	24	29	29	54	45	1,437	842
Pennsylvania	19	38	51	51	----	----	----	----	92	182	7,508	3,823
E. NO. CEN.												
Ohio	37	48	22	30	----	----	----	103	24	31	2,170	421
Indiana	25	17	38	27	902	607	22	89	34	23	955	528
Illinois	21	32	41	41	813	1,241	14	66	15	23	6,933	1,139
Michigan [2]	13	12	33	12	453	429	2	2	338	320	3,564	73
Wisconsin	0	0	13	6	1,026	584	74	98	1,909	1,086	4,316	1,136
W. NO. CEN.												
Minnesota	6	3	3	3	23	12	5	3	2,171	1,120	63	227
Iowa	8	4	5	8	2,192	1,083	1	6	389	192	54	54
Missouri	32	25	13	20	828	644	146	355	18	14	907	662
North Dakota	0	0	0	2	2,658	364	2	12	1,570	215	8	8
South Dakota	30	4	0	2	579	77	2	2	2,104	280	0	3
Nebraska	0	0	17	9	8	2	42	----	160	42	23	29
Kansas	3	1	11	15	324	116	21	29	28	10	382	246
SO. ATL.												
Delaware	39	2	3	1	----	----	----	----	----	----	26	66
Maryland [2]	15	5	14	9	382	124	10	53	3,321	1,077	66	146
Dist. of Col	57	7	7	7	202	25	1	2	154	19	5	25
Virginia	30	16	23	16	2,828	1,509	----	----	472	252	461	461
West Virginia	19	7	12	12	728	271	52	218	35	13	531	73
North Carolina	20	14	31	19	142	97	36	174	2,284	1,563	2,659	787
South Carolina [2]	33	12	9	6	3,226	1,181	481	799	74	27	610	73
Georgia [2]	3	2	3	8	232	140	----	304	254	153	404	----
Florida	15	5	13	8	27	9	1	33	567	188	569	102

See footnotes at end of table.

Cases of certain diseases reported by telegraph by State health officers for the week ended March 4, 1939, rates per 100,000 population (annual basis), and comparison with corresponding week of 1938 and 5-year median—Continued

Division and State	Diphtheria				Influenza				Measles			
	Mar. 4, 1939, rate	Mar. 4, 1939, cases	Mar. 5, 1938, cases	1934–38, median	Mar. 4, 1939, rate	Mar. 4, 1939, cases	Mar. 5, 1938, cases	1934–38, median	Mar. 4, 1939, rate	Mar. 4, 1939, cases	Mar. 5, 1938, cases	1934–38, median
E. SO. CEN.												
Kentucky	12	7	13	16	2,343	1,348	41	80	97	56	602	269
Tennessee	14	8	7	10	257	146	75	215	141	80	725	52
Alabama [3]	14	8	11	19	1,054	599	195	889	401	228	1,117	463
Mississippi [2 3]	10	4	8	4								
W. SO. CEN.												
Arkansas	17	7	11	3	3,654	1,473	184	140	189	76	310	58
Louisiana [3]	19	8	10	15	73	30	7	37	443	183	7	51
Oklahoma	18	9	10	10	672	334	222	244	298	148	58	54
Texas [3]	33	40	47	47	800	965	888	897	273	330	594	538
MOUNTAIN												
Montana	0	0	0	2	1,180	126		29	3,398	363	49	49
Idaho	10	1	1	0	10	1	7	3	806	79	3	28
Wyoming	131	6	0	0	22	1			2,506	119	17	17
Colorado	14	3	15	8	722	150			472	98	620	188
New Mexico	12	1	3	7	704	57	5	8	470	38	60	60
Arizona	61	5	0	1	1,767	144	85	85	380	31	9	37
Utah [3]	10	1	6	0	526	53			1,291	130	281	24
PACIFIC												
Washington	12	4	6	3	25	8	4	3	1,085	352	8	132
Oregon	5	1	0	0	482	97	68	109	298	60	33	116
California	37	45	27	33	41	50	53	202	3,153	3,845	398	564
Total	18	456	606	606	674	14,288	2,798	5,727	643	15,922	41,011	30,806
9 weeks [4]	22	4,939	5,803	5,918	268	51,047	27,416	49,965	477	106,124	242,887	166,268

Division and State	Meningitis, meningococcus				Poliomyelitis				Scarlet fever			
	Mar. 4, 1939, rate	Mar. 4, 1939, cases	Mar. 5, 1938, cases	1934–38, median	Mar. 4, 1939, rate	Mar. 4, 1939, cases	Mar. 5, 1938, cases	1934–38, median	Mar. 4, 1939, rate	Mar. 4, 1939, cases	Mar. 5, 1938, cases	1934–38, median
NEW ENG.												
Maine	0	0	0	0	0	0	0	0	163	27	12	20
New Hampshire	0	0	0	0	0	0	0	0	41	4	33	14
Vermont	0	0	0	0	0	0	0	0	121	9	16	13
Massachusetts	2.4	2	3	3	0	0	0	0	269	229	279	224
Rhode Island	0	0	0	0	0	0	0	0	92	12	18	18
Connecticut	0	0	1	1	0	0	0	0	291	98	90	89
MID. ATL.												
New York	2.4	6	10	13	0.4	1	0	1	255	638	905	948
New Jersey	0	0	3	3	0	0	0	1	233	196	125	182
Pennsylvania	3	6	3	6	0.5	1	1	1	205	404	608	720
E. NO. CEN.												
Ohio	2.3	3	0	9	0	0	0	0	497	646	293	491
Indiana	0	0	2	1	1.5	1	1	0	303	204	198	281
Illinois	0.7	1	3	9	0.7	1	1	1	338	516	699	707
Michigan [3]	0	0	2	2	0	0	1	1	496	469	620	620
Wisconsin	0	0	2	3	0	0	0	0	515	293	170	333

See footnotes at end of table.

Cases of certain diseases reported by telegraph by State health officers for the week ended March 4, 1939, rates per 100,000 population (annual basis), and comparison with corresponding week of 1938 and 5-year median—Continued

Division and State	Meningitis, meningococcus				Poliomyelitis				Scarlet fever			
	Mar. 4, 1939, rate	Mar. 4, 1939, cases	Mar. 5, 1938, cases	1934–38, median	Mar. 4, 1939, rate	Mar. 4, 1939, cases	Mar. 5, 1938, cases	1934–38, median	Mar. 4, 1939, rate	Mar. 4, 1939, cases	Mar. 5, 1938, cases	1934–38, median
W. NO. CEN.												
Minnesota	0	0	2	2	0	0	1	0	215	111	125	149
Iowa	0	0	1	1	0	0	0	0	255	126	257	106
Missouri	2.6	2	1	3	0	0	0	0	161	125	233	219
North Dakota	0	0	0	0	0	0	0	0	110	15	29	50
South Dakota	0	0	1	1	0	0	0	0	173	23	24	24
Nebraska	4	1	18	2	0	0	0	1	157	41	67	66
Kansas	2.8	1	2	2	0	0	0	0	430	154	217	217
SO. ATL.												
Delaware	0	0	0	0	0	0	0	0	0	0	16	16
Maryland [2]	0	0	4	4	0	0	0	0	145	47	73	91
Dist. of Col.	16	2	2	2	0	0	0	0	162	20	25	25
Virginia	0	0	5	5	0	0	1	1	75	40	40	46
West Virginia	0	0	2	2	5	2	1	0	108	40	82	81
North Carolina	2.9	2	5	5	0	0	1	1	47	32	58	44
South Carolina [2]	2.7	1	1	1	0	0	0	0	14	5	4	5
Georgia [2]	0	0	1	1	0	0	0	0	22	13	7	7
Florida	0	0	0	2	0	0	0	1	54	18	8	4
E. SO. CEN.												
Kentucky	5	3	6	6	0	0	0	0	118	68	89	58
Tennessee	1.8	1	6	6	0	0	0	0	93	53	37	28
Alabama [2]	1.8	1	3	2	1.8	1	1	1	37	21	11	11
Mississippi [2 3]	0	0	0	0	0	0	1	0	18	7	9	12
W. SO. CEN.												
Arkansas	0	0	0	1	0	0	0	0	22	9	9	9
Louisiana [2]	0	0	0	1	2.4	1	0	0	15	6	11	12
Oklahoma	0	0	0	3	0	0	1	0	91	45	31	31
Texas [2]	3	4	4	5	0	0	3	1	74	89	127	113
MOUNTAIN												
Montana	0	0	0	1	0	0	0	0	253	27	31	31
Idaho	0	0	0	0	10	1	1	0	184	18	34	16
Wyoming	0	0	0	0	0	0	0	0	44	2	37	37
Colorado	5	1	0	0	0	0	0	0	116	24	46	73
New Mexico	0	0	0	1	12	1	0	0	334	27	30	26
Arizona	25	2	0	0	0	0	1	0	123	10	6	11
Utah [2]	0	0	1	1	0	0	0	0	417	42	54	54
PACIFIC												
Washington	0	0	2	1	0	0	0	0	194	63	51	65
Oregon	0	0	0	0	0	0	0	0	234	47	68	39
California	4	5	5	5	1.6	2	2	3	234	285	212	250
Total	1.8	44	103	154	0.5	12	18	21	215	5,398	6,224	7,153
9 weeks	2.1	481	858	987	0.6	145	192	192	213	48,148	54,300	57,724

See footnotes at end of table.

Cases of certain diseases reported by telegraph by State health officers for the week ended March 4, 1939, rates per 100,000 population (annual basis), and comparison with corresponding week of 1938 and 5-year median—Continued

Division and State	Smallpox				Typhoid and paratyphoid fever				Whooping cough		
	Mar. 4, 1939, rate	Mar. 4, 1939, cases	Mar. 5, 1938, cases	1934-38, median	Mar. 4, 1939, rate	Mar. 4, 1939, cases	Mar. 5, 1938, cases	1934-38, median	Mar. 4, 1939, rate	Mar. 4, 1939, cases	Mar. 5, 1938, cases
NEW ENG.											
Maine	0	0	0	0	6	1	1	1	386	64	89
New Hampshire	0	0	0	0	0	0	0	0	0	0	16
Vermont	0	0	0	0	0	0	0	1	469	35	42
Massachusetts	0	0	0	0	0	0	1	1	269	229	117
Rhode Island	0	0	0	0	0	0	0	0	733	96	29
Connecticut	0	0	0	0	0	0	0	1	229	77	46
MID. ATL.											
New York	0	0	0	0	2	5	2	6	197	491	472
New Jersey	0	0	0	0	2	2	2	2	688	578	177
Pennsylvania	0	0	0	0	2	4	3	3	143	282	285
E. NO. CEN.											
Ohio	17	22	23	1	2	2	2	2	122	159	116
Indiana	128	86	50	3	3	2	1	3	21	14	22
Illinois	10	15	31	6	1	1	4	4	176	269	121
Michigan [1]	14	13	16	1	1	1	6	6	218	206	177
Wisconsin	9	5	4	15	0	0	1	0	480	273	141
W. NO. CEN.											
Minnesota	23	12	28	8	0	0	1	0	68	35	30
Iowa	75	37	24	18	0	0	0	1	38	19	35
Missouri	8	6	46	17	1	1	4	2	62	48	39
North Dakota	22	3	9	8	0	0	0	0	95	13	24
South Dakota	83	11	15	3	0	0	0	0	45	6	17
Nebraska	53	14	9	9	0	0	0	0	11	3	19
Kansas	11	4	28	28	0	0	0	0	61	22	155
SO. ATL.											
Delaware	0	0	0	0	0	0	0	0	30	2	6
Maryland [1]	0	0	0	0	0	0	2	2	71	23	86
Dist. of Col.	0	0	0	0	8	1	0	0	137	17	9
Virginia	0	0	0	0	15	8	1	2	126	67	76
West Virginia	0	0	0	1	16	6	4	3	65	24	70
North Carolina	0	0	1	0	4	3	0	0	308	211	417
South Carolina [2]	0	0	0	0	3	1	0	1	290	106	96
Georgia [2]	0	0	7	1	7	4	5	3	20	12	11
Florida	0	0	0	0	18	6	2	2	93	31	0
E. SO. CEN.											
Kentucky	7	4	30	0	9	5	2	2	17	10	134
Tennessee	12	7	10	0	0	0	0	1	72	41	64
Alabama [1]	0	0	0	0	2	1	0	2	90	51	24
Mississippi [1] [3]	0	0	1	0	8	3	1	1	----	----	----
W. SO. CEN.											
Arkansas	2	1	6	1	2	1	1	1	42	17	52
Louisiana [1]	0	0	0	1	131	54	21	7	2	1	12
Oklahoma	111	55	30	8	2	1	3	3	12	6	41
Texas [1]	21	25	29	7	17	20	7	7	80	96	304
MOUNTAIN											
Montana	28	3	23	8	0	0	0	0	66	7	28
Idaho	163	16	9	4	10	1	0	0	0	0	9
Wyoming	0	0	0	1	22	1	0	0	22	1	19
Colorado	0	0	10	8	10	2	1	2	169	35	8
New Mexico	0	0	0	1	49	4	2	4	556	45	36
Arizona	74	6	0	0	0	0	0	0	233	19	40
Utah [1]	0	0	3	0	0	0	0	0	507	51	28
PACIFIC											
Washington	12	4	64	11	6	2	1	1	99	32	124
Oregon	45	9	16	1	0	0	0	0	65	13	7
California	31	38	12	1	2	3	0	3	133	162	403
Total	16	396	534	233	6	146	81	100	162	3,999	4,273
9 weeks	16	3,597	5,184	1,851	5	1,037	1,067	1,067	171	38,184	36,089

[1] New York City only.
[2] Period ended earlier than Saturday.
[3] Typhus fever, week ended Mar. 4, 1939, 23 cases as follows: South Carolina, 2; Georgia, 4; Alabama, 3; Mississippi, 1; Louisiana, 1; Texas, 12.
[4] The discrepancy of 2 cases of diphtheria in the totals for 6 weeks and 7 weeks (Public Health Reports of Feb. 24 and Mar. 3, pp. 314 and 362) was a correction from 11 to 9 cases in the report for Florida for the week ended Jan. 21, 1939 (Public Health Reports Feb. 3, 1939, p. 193).

SUMMARY OF MONTHLY REPORTS FROM STATES

The following summary of cases reported monthly by States is published weekly and covers only those States from which reports are received during the current week.

State	Meningitis, meningococcus	Diphtheria	Influenza	Malaria	Measles	Pellagra	Poliomyelitis	Scarlet fever	Smallpox	Typhoid and paratyphoid fever
January 1939										
Arizona	2	41	549	1	11	4	0	16	85	3
California	11	142	199	3	7,826	5	4	968	77	19
Hawaii Territory	1	14	135	1	11	----	1	1	0	2
New Hampshire	0	----	----	----	----	----	0	----	0	2
North Dakota	1	10	54	----	1,103	----	0	84	22	2
Ohio	3	149	107	----	101	1	2	2,126	142	19
Utah	1	0	19	----	109	----	0	112	0	1
Virginia	9	96	1,615	1	333	3	2	162	0	15
Wisconsin	1	7	231	----	2,185	----	1	1,154	42	1
February 1939										
Arkansas	3	40	541	74	391	20	2	56	11	11
Connecticut	1	6	86	----	2,159	----	0	412	0	1
Delaware	0	5	----	----	4	----	0	31	0	0
Texas	7	169	3,030	132	572	76	1	405	148	44

January 1939

	Cases
Actinomycosis:	
Hawaii Territory	1
Botulism:	
California	5
Chickenpox:	
Arizona	128
California	3,328
Hawaii Territory	103
North Dakota	104
Ohio	3,033
Utah	632
Virginia	459
Wisconsin	3,140
Conjunctivitis:	
Hawaii Territory	3
Diarrhea:	
Ohio (under 2 yrs., enteritis included)	15
Dysentery:	
Arizona (bacillary)	32
California (amoebic)	10
California (bacillary)	22
Hawaii Territory (amoebic)	5
Virginia (amoebic)	1
Virginia (bacillary)	85
Wisconsin (amoebic)	1
Encephalitis, epidemic or lethargic:	
Arizona	1
California	4
Ohio	2
Utah	1
Wisconsin	1
Food poisoning:	
California	158
German measles:	
Arizona	7
California	126
North Dakota	4
Ohio	21
Utah	10
Granuloma, coccidioidal:	
California	11
Hookworm disease:	
Hawaii Territory	17
Impetigo contagiosa:	
Hawaii Territory	14
Jaundice, infectious:	
California	1

January 1939—Continued

	Cases
Lead poisoning:	
Ohio	16
Leprosy:	
Hawaii Territory	7
Ohio	1
Mumps:	
Arizona	16
California	3,412
Hawaii Territory	129
North Dakota	32
Ohio	1,858
Utah	619
Virginia	313
Wisconsin	563
Ophthalmia neonatorum:	
California	2
Ohi	71
Virginia	3
Puerperal septicemia:	
Ohio	4
Rabies in animals:	
California	149
Septic sore throat:	
California	10
Hawaii Territory	2
North Dakota	1
Ohio	143
Virginia	127
Wisconsin	14
Tetanus:	
California	3
Hawaii Territory	3
Virginia	1
Trachoma:	
Arizona	43
California	23
Hawaii Territory	3
Trichinosis:	
Arizona	8
California	5
Ohio	1
Tularaemia:	
California	1
Ohio	29
Virginia	17
Wisconsin	1
Typhus fever:	
California	5
Hawaii Territory	2

January 1939—Continued

	Cases
Undulant fever:	
California	9
Ohio	10
Virginia	1
Wisconsin	3
Vincent's infection:	
North Dakota	6
Whooping cough:	
Arizona	36
California	424
Hawaii Territory	47
North Dakota	76
Ohio	918
Utah	56
Virginia	220
Wisconsin	1,321

February 1939

	Cases
Anthrax:	
Delaware	3
Chickenpox:	
Arkansas	249
Connecticut	438
Delaware	149
Texas	1,635
Conjunctivitis, infectious:	
Connecticut	7
Dengue:	
Texas	3
Dysentery:	
Arkansas (amoebic)	3
Arkansas (bacillary)	1
Connecticut (bacillary)	3
Texas (bacillary)	30
Encephalitis, epidemic or lethargic:	
Texas	4
Hookworm disease:	
Arkansas	1
Leprosy:	
Texas	2
Mumps:	
Arkansas	21
Connecticut	306
Delaware	123
Texas	162
Ophthalmia neonatorum:	
Arkansas	2
Texas	3
Puerperal septicemia:	
Arkansas	2

February 1939—Continued

Rabies in animals:	Cases
Arkansas	31
Connecticut	4
Delaware	1
Texas	12
Relapsing fever:	
Texas	10
Septic sore throat:	
Arkansas	47
Connecticut	19

February 1939—Continued

Trachoma:	Cases
Arkansas	4
Connecticut	1
Texas	30
Trichinosis:	
Connecticut	2
Tularaemia:	
Arkansas	2
Texas	8
Typhus fever:	
Texas	19

February 1939—Continued

Undulant fever:	Cases
Arkansas	4
Connecticut	6
Texas	12
Whooping cough:	
Arkansas	62
Connecticut	320
Delaware	18
Texas	401

WEEKLY REPORTS FROM CITIES

City reports for week ended February 25, 1939

This table summarizes the reports received weekly from a selected list of 140 cities for the purpose of showing a cross section of the current urban incidence of the communicable diseases listed in the table.

State and city	Diphtheria cases	Influenza		Measles cases	Pneumonia deaths	Scarlet fever cases	Smallpox cases	Tuberculosis deaths	Typhoid fever cases	Whooping cough cases	Deaths, all causes
		Cases	Deaths								
Data for 90 cities:											
5-year average	188	839	139	6,938	994	2,303	24	412	18	1,247	----
Current week	156	1,339	159	4,417	943	1,546	82	371	47	1,264	----
Maine:											
Portland	0	----	0	0	2	2	0	0	0	4	28
New Hampshire:											
Concord	0	----	0	0	3	0	0	1	0	0	10
Manchester	0	----	0	0	2	3	0	0	0	0	15
Nashua	0	----	0	0	0	0	0	0	0	0	4
Vermont:											
Barre	0	----	0	0	1	0	0	0	0	1	3
Burlington	0	----	0	1	0	0	0	0	0	0	9
Rutland	0	----	0	0	0	0	0	0	0	0	2
Massachusetts:											
Boston	0	----	2	167	30	57	0	9	0	21	252
Fall River	0	----	0	1	6	3	0	1	0	2	33
Springfield	0	----	0	21	4	2	0	3	0	4	49
Worcester	0	----	0	4	5	18	0	1	0	53	56
Rhode Island:											
Pawtucket	0	----	0	2	0	3	0	0	0	5	17
Providence	0	4	0	9	1	11	0	2	0	80	46
Connecticut:											
Bridgeport	0	1	1	0	2	5	0	2	0	4	----
Hartford	0	----	0	162	4	10	0	0	0	5	41
New Haven	2	4	1	70	6	2	0	2	0	7	63
New York:											
Buffalo	1	----	1	83	10	77	0	8	0	23	123
New York	28	101	12	80	150	233	0	75	2	151	1,684
Rochester	0	4	0	106	3	31	0	0	0	16	73
Syracuse	0	----	0	110	5	26	0	2	0	38	53
New Jersey:											
Camden	4	1	2	0	2	3	0	0	0	0	39
Newark	1	4	1	5	10	46	0	5	0	93	126
Trenton	0	1	0	0	6	5	0	7	0	5	44
Pennsylvania:											
Philadelphia	5	17	10	29	46	76	0	24	2	106	592
Pittsburgh	2	18	8	3	19	48	0	10	0	16	212
Reading	6	----	0	2	2	1	0	0	0	0	36
Scranton	0	----	----	3	----	30	0	----	9	9	----
Ohio:											
Cincinnati	7	27	2	0	22	34	0	5	0	3	148
Cleveland	2	345	3	5	24	57	0	9	0	44	239
Columbus	0	1	1	4	6	4	0	2	0	4	55
Toledo	0	1	1	2	8	20	0	4	0	17	83
Indiana:											
Anderson	0	----	0	0	0	6	0	1	0	1	14
Fort Wayne	1	----	0	1	2	10	0	0	0	0	26
Indianapolis	1	----	5	2	29	38	67	2	0	9	132
Muncie	0	----	0	0	5	4	1	0	0	0	17
South Bend	0	----	0	1	4	4	0	1	0	6	22
Terre Haute	0	----	2	1	2	1	0	0	0	0	27
Illinois:											
Alton	0	----	0	0	0	0	0	0	0	0	5
Chicago	15	351	34	9	135	178	1	48	0	128	1,092
Elgin	0	----	0	0	3	10	0	0	0	1	16
Moline	0	56	0	0	0	0	0	0	0	4	6
Springfield	0	2	1	0	2	3	0	0	0	6	14

City reports for week ended February 25, 1939—Continued

State and city	Diphtheria cases	Influenza		Measles cases	Pneumonia deaths	Scarlet fever cases	Smallpox cases	Tuberculosis deaths	Typhoid fever cases	Whooping cough cases	Deaths, all causes
		Cases	Deaths								
Michigan:											
Detroit	16	82	15	9	67	122	0	19	1	96	398
Flint	0	----	1	156	3	18	0	1	0	0	31
Grand Rapids	0	82	2	2	3	16	0	0	0	2	47
Wisconsin:											
Kenosha	0	----	0	0	2	4	0	0	0	20	14
Madison	0	----	0	1	2	7	0	0	0	7	23
Milwaukee	0	57	1	3	6	71	0	3	0	94	123
Racine	0	----	0	3	0	5	0	0	0	1	16
Superior	0	----	0	9	1	2	0	0	0	0	9
Minnesota:											
Duluth	0	20	----	3	1	4	0	1	0	5	19
Minneapolis	2	----	4	266	2	23	6	1	0	28	129
St. Paul	1	----	0	446	4	16	0	3	0	6	64
Iowa:											
Cedar Rapids	0	----	----	1	----	0	0	----	0	3	----
Davenport	0	----	----	0	----	6	2	----	0	0	----
Des Moines	0	----	0	1	0	23	1	0	0	0	34
Sioux City	0	----	----	26	----	1	0	----	0	2	----
Waterloo	2	----	----	1	----	13	1	----	0	1	----
Missouri:											
Kansas City	0	1	6	1	26	13	0	5	0	1	137
St. Joseph	0	----	0	0	3	3	0	0	5	3	13
St. Louis	2	14	1	0	12	27	1	8	0	39	208
North Dakota:											
Fargo	1	----	0	1	1	7	0	0	0	0	19
Grand Forks	0	----	----	1	----	0	0	----	0	2	----
Minot	0	----	0	21	0	0	0	0	0	0	4
South Dakota:											
Aberdeen	0	----	----	2	----	1	1	----	0	0	----
Sioux Falls	2	----	0	23	0	2	0	0	0	0	9
Nebraska:											
Lincoln	0	----	----	10	----	0	0	----	0	3	----
Omaha	0	----	0	5	4	8	0	2	0	0	50
Kansas:											
Lawrence	0	41	0	0	1	1	0	0	0	0	7
Topeka	0	----	0	0	4	2	0	0	0	0	23
Wichita	0	----	0	0	3	4	0	1	0	0	22
Delaware:											
Wilmington	1	----	0	0	6	2	0	0	0	0	37
Maryland:											
Baltimore	3	79	9	1,010	44	27	0	9	0	7	200
Cumberland	0	3	0	0	0	0	0	0	0	0	14
Frederick	0	----	0	0	0	0	0	0	0	1	5
Dist. of Columbia:											
Washington	7	25	2	19	17	20	0	10	1	17	235
Virginia:											
Lynchburg	1	----	0	79	4	2	0	1	0	7	20
Norfolk	0	211	1	3	5	5	0	1	0	3	36
Richmond	0	----	3	25	5	3	0	3	0	0	54
Roanoke	1	----	0	0	2	1	0	0	0	3	20
West Virginia:											
Charleston	0	2	0	0	1	0	0	0	0	0	10
Huntington	1	----	----	0	----	0	0	----	0	0	----
Wheeling	0	----	0	2	3	1	0	2	0	4	23
North Carolina:											
Gastonia	1	----	0	1	0	0	0	1	0	0	----
Raleigh	0	----	0	1	4	3	0	2	0	0	30
Wilmington	0	----	0	0	1	0	0	0	1	10	9
Winston-Salem	1	----	1	214	1	0	0	1	0	1	13
South Carolina:											
Charleston	0	13	0	0	0	3	0	2	1	0	15
Florence	1	----	0	0	4	0	0	1	0	0	15
Greenville	0	----	0	0	1	0	0	0	0	5	8
Georgia:											
Atlanta	0	22	1	1	9	8	0	6	0	0	92
Brunswick	0	----	0	59	2	0	0	0	0	0	6
Savannah	0	17	0	0	3	0	0	0	0	3	35
Florida:											
Miami	0	4	1	1	3	3	0	0	0	3	56
Tampa	0	1	1	92	3	1	0	1	0	0	23
Kentucky:											
Ashland	0	2	2	0	2	0	0	0	0	0	9
Covington	1	3	0	0	4	12	0	2	0	0	17
Lexington	1	----	0	2	3	5	0	2	0	0	19
Louisville	1	35	3	0	10	13	0	3	0	3	73

City reports for week ended February 25, 1939—Continued

State and city	Diphtheria cases	Influenza		Measles cases	Pneumonia deaths	Scarlet fever cases	Smallpox cases	Tuberculosis deaths	Typhoid fever cases	Whooping cough cases	Deaths, all causes
		Cases	Deaths								
Tennessee:											
Knoxville	1		0	0	1	3	0	0	0	0	31
Memphis	0	3	1	0	15	5	0	3	0	9	79
Nashville	0		3	0	2	6	0	1	0	1	42
Alabama:											
Birmingham	0	6	1	0	5	2	0	3	0	0	61
Mobile	0		3	0	5	1	0	1	0	0	33
Montgomery	0	10		8		0	0		0	0	
Arkansas:											
Fort Smith	0			1		1	0		0	0	
Little Rock	0			0	3	0	0		0	0	
Louisiana:											
Lake Charles	1		0	63	2	0	0	0	0	0	15
New Orleans	15	7	2	49	17	7	0	9	31	10	156
Shreveport	1		0	5	10	6	0	0	0	0	52
Oklahoma:											
Oklahoma City	0	14	0	2	6	11	0	1	0	0	50
Tulsa	1			2		7	0		0	0	
Texas:											
Dallas	1	2	1	10	11	5	2	1	0	3	60
Fort Worth	1	14	0	1	6	7	3	1	0	0	27
Galveston	0		0	0	2	0	0	0	1	0	13
Houston	2	1	1	16	15	6	0	4	0	1	99
San Antonio	2	3	1	6	7	5	0	11	0	0	80
Montana:											
Billings	2		0	10	2	0	0	0	0	0	10
Great Falls	0		0	2	3	0	0	0	0	0	15
Helena	0		0	119	0	1	0	1	0	0	9
Missoula	0	1	0	26	0	0	1	0	0	0	4
Idaho:											
Boise	0		0	0	0	1	0	0	0	0	4
Colorado:											
Colorado Springs	0		0	61	0	2	0	0	0	12	11
Denver	6		6	5	5	8	0	1	0	28	95
Pueblo	0		0	6	3	3	0	0	0	5	13
New Mexico:											
Albuquerque	0		0	0	1	0	0	0	0	5	12
Utah:											
Salt Lake City	0		1	2	1	14	0	1	0	2	38
Washington:											
Seattle	0		2	53	9	7	0	2	0	6	109
Spokane	0		0	62	4	3	0	0	1	0	37
Tacoma	0		0	0	0	2	0	0	0	0	29
Oregon:											
Portland	1	1	1	1	5	6	3	1	0	2	90
Salem	0	3		2		1	0		0	0	
California:											
Los Angeles	14	16	1	213	21	47	2	17	1	18	369
Sacramento	1		0	135	3	0	2	3	0	1	33
San Francisco	1	1	3	412	10	16	0	13	0	3	212

State and city	Meningitis, meningococcus		Poliomyelitis cases
	Cases	Deaths	
Massachusetts:			
Boston	1	1	0
Rhode Island:			
Pawtucket	1	1	0
Providence	1	0	0
New York:			
Buffalo	2	0	0
New York	2	2	0
Pennsylvania:			
Philadelphia	1	0	0
Pittsburgh	1	1	0
Ohio:			
Cincinnati	1	0	0
Illinois:			
Chicago	1	0	0
Maryland:			
Baltimore	1	0	0
District of Columbia:			
Washington	1	1	0
Florida:			
Tampa	0	0	1
Alabama:			
Birmingham	1	0	0
Louisiana:			
New Orleans	2	0	0
Shreveport	0	1	0
Texas:			
Dallas	1	0	0
Houston	1	0	0

Encephalitis, epidemic or lethargic.—Cases: New York, 1.
Pellagra.—Cases: Wilmington, N. C., 1; Atlanta, 7; Savannah, 1; Dallas, 1; Los Angeles, 1.
Typhus fever.—Cases: Charleston, S. C., 2; Houston, 1; San Antonio, 2.

FOREIGN AND INSULAR

CANADA

Provinces—Communicable diseases—Week ended January 28, 1939.—During the week ended January 28, 1939, cases of certain communicable diseases were reported by the Department of Pensions and National Health of Canada as follows:

Disease	Prince Edward Island	Nova Scotia	New Brunswick	Quebec	Ontario	Manitoba	Saskatchewan	Alberta	British Columbia	Total
Cerebrospinal meningitis					1					1
Chickenpox			25	206	384	25	33	10	81	764
Diphtheria		5	3	29	2	3	3			45
Dysentery									2	2
Influenza					164				26	190
Measles		3		55	933	27	5	1	2	1,026
Mumps				116	24	18		8	1	167
Pneumonia		3			29		1		10	43
Scarlet fever		4	14	104	195	33	31	31	8	420
Smallpox							1			1
Trachoma						1			1	2
Tuberculosis		1	8	22	50	24	4	1	13	123
Typhoid and paratyphoid fever				5		1	1			7
Whooping cough				286	343	16	5	1	73	724

FINLAND

Communicable diseases—January 1939.—During the month of January 1939, cases of certain communicable diseases were reported in Finland as follows:

Disease	Cases	Disease	Cases
Diphtheria	304	Scarlet fever	741
Influenza	3,514	Typhoid fever	6
Paratyphoid fever	12	Undulant fever	2
Poliomyelitis	18		

LATVIA

Notifiable diseases—October–December 1938.—During the months of October, November, and December 1938, cases of certain notifiable diseases were reported in Latvia as follows:

Disease	October	November	December	Disease	October	November	December
Botulism		1	1	Poliomyelitis	154	64	26
Cerebrospinal meningitis	11	6	4	Puerperal septicemia	11	3	5
Diphtheria	171	244	240	Scarlet fever	333	455	471
Erysipelas	47	36	40	Tetanus	3	2	2
Influenza	55	54	49	Trachoma	64	48	38
Lead poisoning	1	3	6	Tuberculosis	214	240	212
Lethargic encephalitis		1		Typhoid fever	54	53	82
Measles	17	16	76	Typhus fever		1	
Mumps	52	145	347	Whooping cough	79	51	50
Paratyphoid fever	12	10	6				

YUGOSLAVIA

Communicable diseases—4 weeks ended January 29, 1939.—During the 4 weeks ended January 29, 1939, certain communicable diseases were reported in Yugoslavia as follows:

Disease	Cases	Deaths	Disease	Cases	Deaths
Anthrax	23	4	Paratyphoid fever	5	
Cerebrospinal meningitis	48	10	Poliomyelitis	2	1
Diphtheria and croup	585	43	Scarlet fever	258	12
Dysentery	8		Sepsis	11	1
Erysipelas	159	5	Tetanus	11	4
Favus	9		Typhoid fever	316	22
Lethargic encephalitis	3	2	Typhus fever	22	2

CHOLERA, PLAGUE, SMALLPOX, TYPHUS FEVER, AND YELLOW FEVER

NOTE.—A table giving current information of the world prevalence of quarantinable diseases appeared in the PUBLIC HEALTH REPORTS for February 24, 1939, pages 322-333. A similar cumulative table will appear in future issues of the PUBLIC HEALTH REPORTS for the last Friday of each month.

Plague

Egypt—Asyut Province.—During the week ended February 25, 1939, 26 cases of plague were reported in Asyut Province, Egypt.

Peru.—During the month of January 1939, plague was reported in Peru as follows: Lambayeque Department, 2 cases, 1 death; Libertad Department, 11 cases, 6 deaths; Lima Department, 1 case.

Yellow Fever

Brazil—Espirito Santo State.—Yellow fever has been reported in Espirito Santo State, Brazil, as follows: Alegre, January 27, 1939, 1 death; Sabino Pessoa, January 27, 1939, 1 death; Sao Felipe, January 25, 1939, 1 death.

Niger Territory—Say.—On February 23, 1939, 1 suspected case of yellow fever was reported in Say, Niger Territory.

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